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PRESIDENT'S ADDRESS*

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SOME time ago I decided to address you on a phase of medical economics. Along came the National Health Conference in Washington, and I contemplated a bit. I wrote one of our members that I did not believe that the American Medical Association and the Inter-Departmental Committee to Coordinate Health and Welfare Activities were really very far apart in their ideas. As I started to get my thoughts into some semblance of logical order a telegram arrived with the call for a special session of the House of Delegates of the American Medical Association. I put off my paper until such time as I should return from Chicago.

Fortunately, there is not much to write about. It has been clearly proved, in spite of all the hard feelings and of the epithets and charges bandied back and forth, that the ideas of the American Medical Association do not differ widely from the utopian concept of the authorities in Washington. The misunderstandings arose from two sources: first, a lack of a clear understanding of the methods by which the American Medical Association functions and, second, the fact that the Association has couched all its resolutions and rules in negative rather than positive phraseology. It is a very easy matter to call the American Medical Association arbitrary, dictatorial, unco-operative, reactionary and even monopolistic.

The Association, according to Article I of the constitution, is a federacy. The *American Dictionary and Cyclopedia* defines a federacy as "a federation or union of several states under one central authority, consisting of delegates from each state in matters of general policy, but self-governing in local matters." Please note that the delegates are the central authority. Nothing can be done unless the delegates say so. The president, general manager, editor and trustees, the so-called general officers, have less authority in the Association than in any other organization with which I am familiar. Chapter XVII of the by-laws states: "No memorial, resolution or *opinion* [italics mine] of any character whatever shall be issued in the name of the American Medical Association unless it has been approved by the House of Delegates."

The members of the American Medical Association present at the Washington conference could not accept, reject or endorse the committee report because the House of Delegates had not yet passed on it. Any expression of the members present was that of an individual and not of organized medicine. The Association is the only organization so constituted that it can speak for physicians of the United States. Its membership is over one hundred and ten thousand. The House of Delegates is made up of one hundred and seventy-four duly elected representatives, of whom one hundred and sixty-five were present at the recent special session held in Chicago.

This was the third special session in the history of the American Medical Association. The first was held when the United States entered the World War, and the second to consider the Social Security Act.

Let us suppose that a small group of doctors in Massachusetts desires the American Medical Association to take some action concerning a special matter. Proceedings should originate in the county medical society. If that society decides that it is solidly behind our hypothetical group of doctors, it will instruct its councilors to the state society to further the matter. The state society, having taken favorable action, will so instruct its delegates to the American Medical Association. In Massa-

*Read at the annual meeting of the New England Surgical Society, Boston, September 30, 1938.

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chusetts we have several doctors who have appeared in the public print as champions of this or that and as severe critics of the American Medical Association. To the best of my knowledge and belief, these gentlemen have never directly presented their ideas for approval by their county societies or the Massachusetts Medical Society.

In the House of Delegates of the Association, the Massachusetts delegation is exceeded numerically only by those of three other states. The New England delegation, which is made up of men who have always worked most harmoniously together, is exceeded numerically only by the New York delegation. New England should and does have some weight in the House of Delegates. In addition to our delegates we have one of the nine trustees and membership in several of the standing committees and councils. If we ever wish for something, let us go after it in an orderly manner.

The Association, through its numerous committees and councils, has compiled a vast array of authentic facts and figures. It knows that there are more than thirty-five hundred plans for the care of the medically indigent, which are operating in the United States today. It knows the advantages and disadvantages of each and every one of these plans and of the plans in operation in other countries. It knows how long these plans have been in operation and whether sufficient time has elapsed to try them out thoroughly. It knows how many hospitals there are in the United States; where they are located; how many beds they have; what their physical and professional equipment is. These facts and many more covering every phase of medical activity are available. Over a year ago all these data were freely offered to any governmental agency which might wish to avail itself of it. No one seemed to wish. In July of this year the Inter-Departmental Committee, appointed by the President, made its report at the National Health Conference. This report, together with the recommendations of a subcommittee, the Technical Committee on Medical Care, was evolved without the aid of organized medicine, whose cordial co-operation had been freely offered. The American Medical Association was criticized because certain individuals did not endorse the recommendations of the technical committee or approve the actions. Earlier in this paper I have shown that no one had the power or authority to do these things. The House of Delegates had first to pass on this report before anyone could act for organized medicine. Under ordinary circumstances the House of Delegates would not have met until the St. Louis session in June, 1939. Because it seemed probable that some legislation, the outcome of the conference, will be introduced at the next session of Congress, a special session was called to formulate the position of organized medicine on a more or less well-defined proposition.

The technical committee's report was divided into five parts, each covering a certain situation and containing a definite recommendation. At the special session, five committees were appointed, each to deal with one of the five divisions of the Washington report. After the deliberations of these five committees were concluded, the five chairmen met as a supercommittee to draft the final report submitted to the House of Delegates.

* * *

I shall present briefly the five recommendations proposed at the Washington conference and, with each, the stand taken by the American Medical Association,* with a word of explanation of my own.

Recommendation I

The technical committee recommended expansion of the existing federal-state co-operative programs under Public Health Services and Maternal and Child Health Services of the Social Security Act. This recommendation goes into further details concerning the expansion of Public Health Services, as they pertain to the control of tuberculosis, venereal diseases, cancer, pneumonia, and so forth, and of Maternal and Child Health Services, the latter including the care of crippled children.

The resolution of the House of Delegates in considering this recommendation reads as follows:

> Under Recommendation I on Expansion of Public Health Services: (1) Your committee recommends the establishment of a federal department of health with a secretary who shall be a doctor of medicine and a member of the President's Cabinet. (2) The general principles outlined by the Technical Committee for the expansion of public health and maternal and child health services are approved and the American Medical Association definitely seeks to co-operate in developing efficient and economical ways and means of putting into effect this recommendation. (3) Any expenditures made for the expansion of public health and maternal and child health services should not include the treatment of disease except so far as this cannot be successfully accomplished through the private practitioner.

For many years the American Medical Association has advocated a national Department of Health with a cabinet officer at its head. Within recent years we have had two physicians as cabinet officers, so one is justified in believing that adequate material is at hand.

*Proceedings of the Special Session. J. A. M. A. 111:1215, 1938.

Recommendation II

The technical committee recommended federal grants-in-aid for the construction of needed hospitals and similar facilities, and special grants on a diminishing basis toward defraying the operating costs of these new institutions in the first three years of their existence. The comment of the House of Delegates is as follows:

> Under Recommendation II on Expansion of Hospital Facilities: Your committee favors the expansion of general hospital facilities where need exists. The hospital situation would indicate that there is at present greater need for the use of existing hospital facilities than for additional hospitals.
>
> Your committee heartily recommends the approval of the recommendation of the technical committee stressing the use of existing hospital facilities. The stability and efficiency of many existing church and voluntary hospitals could be assured by the payment to them of the costs of the necessary hospitalization of the medically indigent.

You will note that the conference report says nothing about helping to support existing hospitals and only advocates helping new government-built ones for three years.

Recommendation III

The technical committee recommended federal grants-in-aid to the states for assisting in the provision of a medical-care program for recipients of public assistance and other medically needy persons.

The answer reads as follows:

> Under Recommendation III on Medical Care for the Medically Needy: Your committee advocates recognition of the principle that the complete medical care of the indigent is a responsibility of the community, medical and allied professions, and that such care should be organized by local governmental units and supported by tax funds.
>
> Since the indigent now constitute a large group in the population, your committee recognizes that the necessity for state aid for medical care may arise in poorer communities and the federal government may need to provide funds when the state is unable to meet these emergencies.
>
> Reports of the Bureau of the Census, of the United States Public Health Service and of life insurance companies show that great progress has been made in the United States in the reduction of morbidity and mortality among all classes of people. This reflects the good quality of medical care now provided. Your committee wishes to see continued and improved the methods and practices which have brought us to this present high plane.
>
> Your committee wishes to see established well coordinated programs in the various states in the nation, for improvement of food, housing and the other environmental conditions which have the greatest influence on the health of our citizens. Your committee wishes also to see established a definite and far-reaching public health program for the education and information of all the people in order that they may take advantage of the present medical service available in this country.
>
> In the face of the vanishing support of philanthropy, the medical profession as a whole will welcome the appropriation of funds to provide medical care for the medically needy, provided, first, that the public welfare administrative procedures are simplified and coordinated; and, second, that the provision of medical services is arranged by responsible *local* [italics mine] public officials in co-operation with the *local* [italics mine] medical profession and its allied groups.
>
> Your committee feels that in each state a system should be developed to meet the recommendation of the National Health Conference in conformity with its suggestion that "The role of the federal government should be principally that of giving financial and technical aid to the states in their development of sound programs through procedures largely of their own choice."

The program suggested by the House of Delegates goes into more detail than that recommended by the technical committee, and points a warning finger.

Recommendation IV

The technical committee recommended the "consideration of a comprehensive program designed to increase and improve the medical services for the entire population."

The comment reads as follows:

> Under Recommendation IV on a General Program of Medical Care: Your committee approves the principle of hospital service insurance which is being widely adopted throughout the country. It is susceptible of great expansion along sound lines, and your committee particularly recommends it as a community project. Experience in the operation of hospital service insurance or group hospitalization plans has demonstrated that these plans should confine themselves to provision of hospital facilities and should *not* [italics mine] include any type of medical care.
>
> Your committee recognizes that health needs and means to supply such needs vary throughout the United States. Studies indicate that health needs are not identical in different localities but that they usually depend on local conditions and therefore are primarily local problems. Your committee therefore encourages county or district medical societies, with the approval of the state medical society of which each is a component part, to develop appropriate means to meet their local requirements.
>
> In addition to insurance for hospitalization your committee believes it is practicable to develop cash indemnity insurance plans to cover, in whole or in part, the costs of emergency or prolonged illness. Agencies set up to provide such insurance should comply with state statutes and regulations to insure their soundness and financial responsibility and have the approval of the county and state medical societies under which they operate.
>
> Your committee is *not* [italics mine] willing to foster any system of compulsory health insurance. Your committee is convinced that it is a complicated, bureaucratic system which has no place in a democratic state. It would undoubtedly set up a far-reaching tax system

with great increase in the cost of government. That it would lend itself to political control and manipulation there is no doubt.

Your committee recognizes the soundness of the principles of workmen's compensation laws and recommends the expansion of such legislation to provide for meeting the costs of illness sustained as a result of employment in industry.

Your committee repeats its conviction that voluntary indemnity insurance may assist many income groups to finance their sickness costs without subsidy. Further development of group hospitalization and establishment of insurance plans on the indemnity principle to cover the cost of illness will assist in solution of these problems.

Organized medicine is opposed to compulsory health insurance. It is triple compulsion: the individual is compelled to take it; he is compelled to take a certain doctor; and the doctor is compelled to administer the service.

Recommendation V

The technical committee recommended that action be taken toward the development of a program to provide disability compensation.

The comment of the House of Delegates reads as follows:

Under Recommendation V on Insurance Against Loss of Wages During Sickness: In essence, the recommendation deals with compensation of loss of wages during sickness. Your committee unreservedly endorses this principle, as it has distinct influence toward recovery and tends to reduce permanent disability. It is, however, in the interest of good medical care that the attending physician be relieved of the duty of certification of illness and of recovery, which function should be performed by a qualified medical employee of the disbursing agency.

You will note that the House of Delegates is firmly opposed to the idea of having the family physician certify as to disability.

In addition to passing the above five recommendations, the speaker of the House of Delegates appointed a committee of nine physicians "to confer and consult with the proper federal representatives relative to the proposed National Health Program."

* * *

It seems to me that the special session accomplished a great deal and more than justified the expenditure of the time and money involved. The criticism of the American Medical Association, which has emanated from many sources, should be stilled. The Association, as in the past, has shown a commendable desire to co-operate for the common good, while safeguarding the rights of all. It has in no way about-faced or changed its well-known ideals. It has affirmed in positive phraseology the principles evolved through many years of careful study and practice. It accepts in principle, as it has before, the recommendations of the Washington conference. It does foresee the dangers and difficulties inherent in proposed legislation, and firmly places its finger on the troublesome spots.

The controversial problems have not been definitely settled, but a distinct and favorable attempt toward their solution has been inaugurated. In the debates, conferences and differences that are bound to come, remember one thing, you represent the American Medical Association — a true democracy. If you work together with a clear understanding of the principles and methods involved, medicine can go on, as it has in the past, to a brilliant future of accomplishment — a future dedicated to the alleviation of pain and suffering without the thoughts of personal gain.

146 Chestnut Street.

REPORTED GONORRHEA AND SYPHILIS IN MASSACHUSETTS, 1930 - 1937*

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BOSTON

The Massachusetts Department of Public Health is greatly indebted to the 3028 physicians (45.2 per cent of the 6700 physicians in the State) who have reported 10,466 cases of syphilis and 26,567 cases of gonorrhea during the last eight years. This service rendered by the physician not only has made possible morbidity analyses, but has also provided the evidence with which to obtain funds for the continued provision of important services to the physician and his patients, such as blood tests for syphilis, free arsenicals and bismuth, examinations of smears for gonorrhea, instructive literature, postgraduate medical education (in co-operation with the Massachusetts Medical Society) and other related services. The department appreciates the physicians' co-operation and hopes it will continue.

HENRY D. CHADWICK, M.D.,
Commissioner of Public Health.

SINCE 1930 all forms and stages of syphilis and gonorrhea have been reported directly to the Massachusetts Department of Public Health. A large body of data has accumulated, and an analysis of this data is now presented. Admittedly a small section of the true total of reportable infections, it nevertheless discloses certain trends and relations which can be studied. In 1930 and 1931 similar reports[1, 2] were prepared.

In the eight years 1930-1937 inclusive, 91,347 reports of syphilis and of gonorrhea were received from 3028 physicians, 28 clinics and about 180 hospitals and institutions. It is gratifying to note that since 1930 the number of physicians reporting in any one year has increased from 1082 in 1930 to 1507 in 1937. A study of reports made by physicians receiving arsenicals or bismuth free from the Commonwealth shows that of 602 ordering in 1937, 521 (86.5 per cent) have reported syphilis at some time since 1930. This is an encouraging response to an important service. Some of the 13.5 per cent who have not reported ordered arsenicals in anticipation of cases requiring treatment, and a few others have been denied further supplies because of repeated failure to report their cases.

Of the total reports, 39,159 represent syphilis in all stages and 52,188 gonorrhea. Table 1 indicates the trend of reporting since 1930, including both total reports and sources of reports.

Reports from physicians and clinics are not entirely comparable, since less than half the physicians treating syphilis and gonorrhea report their cases whereas clinics report most of theirs. In comparing figures from clinics and physicians, therefore, this fact must be constantly borne in mind in order to interpret reasonably what may superficially appear to be disproportionate percentages.

There has been a considerable increase of reports of syphilis from physicians during the last few years, reflecting, perhaps, the increased state

Table 1. *Sources of Reports of Gonorrhea and Syphilis (February 1, 1930, to December 31, 1937).*

Year	Total Cases Reported	Physicians			Clinics		Institutions	
		Number Reporting	Cases Reported	Percentage of Total Reports	Cases Reported	Percentage of Total Reports	Cases Reported	Percentage of Total Reports
Gonorrhea*								
1930	6974	886	3789	54.3	—§		—§	
1931	7199	886	3844	53.4	2863	39.8	492	6.8
1932	6735	846	3179	47.2	2925	43.4	631	9.4
1933	6597	824	3195	48.4	2872	43.6	530	8.0
1934	6522	845	3153	48.4	2818	43.2	551	8.4
1935	6189	876	3001	48.5	2631	42.5	557	9.0
1936	6096	958	3266	53.6	2279	37.4	551	9.0
1937	5856	988	3140	53.6	2209	37.7	507	8.7
Syphilis†								
1930	4199	474	1105	26.3	—§		—§	
1931	4439	516	1143	25.8	2641	59.5	655	14.7
1932	4527	442	877	19.4	2779	61.4	871	19.2
1933	4471	428	913	20.4	2867	64.2	691	15.4
1934	4464	460	918	20.6	2937	65.8	609	13.6
1935	5317	703	1472	27.7	2783	52.3	1062	20.0
1936	5526	906	1935	35.0	2492	45.1	1099	19.9
1937	6207	961	2103	33.9	2788	44.9	1316	21.2

*Punch-card error of 20 in grand total of 52,188.
†Punch-card error of 9 in grand total of 39,159.
§Clinics and institutions not separated in 1930.

*From the Massachusetts Department of Public Health.
†Epidemiologist, Massachusetts Department of Public Health.

and national publicity. Institutions, too, have reported more cases with each year, the increase in 1937 being due partly to better reporting and partly to the new law governing treatment. Clinics have maintained a relatively stable infected group, their reports of syphilis varying only slightly since 1932. The reports of total cases of gonorrhea have shown a steady decline since 1931. The cause of this definite downward trend is not clear and cannot be ascribed to sulfanilamide therapy, for the decline is evident before the advent of this new drug. The figures for total cases of reported syphilis indicate an increase which is coincident with the recent flood of publicity, and represents an increase in late cases, due to improved reporting and to more routine blood-testing, rather than in early cases, which have maintained a relatively constant level since 1933.

Table 2. *Reported Early (Primary and Secondary) Syphilis and Late Syphilis.*

DIAGNOSIS AND SEX	1930	1931	1932	1933	1934	1935	1936	1937
Early syphilis:								
Male	886	887	814	754	660	700	761	724
Female	635	584	591	481	412	457	479	475
Total	1521	1471	1405	1235	1072	1157	1240	1199
Late syphilis:								
Male	1499	1577	1661	1665	1795	2214	2262	2605
Female	943	1053	1146	1238	1308	1582	1730	2054
Total	2442	2630	2807	2903	3103	3796	3992	4659

During the period 1930-1937, reported early syphilis declined 18.3 per cent in males and 25.2 per cent in females; reported late syphilis increased 73.6 per cent in males and 107.2 per cent in females. It is apparent that the decline in reported early syphilis and the increase in reported late syphilis have been much more rapid in females than in males. The cause of this phenomenon is not clear, unless it be that the increased professional and public interest in the genitoinfections has aroused on the one hand the curiosity of physicians as to the actual extent of syphilis in women, leading to more pertinent history-taking and routine blood-testing, and on the other hand a more active response of women than men to public education.

It is interesting that whereas reported late cases have increased markedly during the last three years, early cases during the same period have remained at a fairly constant level, a level which would undoubtedly have been lower had not the energetic propagandizing of the public and medical profession produced such a salutary effect on reporting. Had reporting continued at an average rate, the decline in gross reports of early syphilis evident from 1930 to 1934 would most likely have continued.

Table 3 shows the trend of the rate of early syphilis since 1930. If the rate is computed on the basis of average reporting and the number of early cases estimated on the basis of percentage (according to year) of average reporting, a relative rate is obtained which tends to correct for abnormal reporting in any one year.

Table 3. *Reported Early (Primary and Secondary) Syphilis, Expressed as Rate per 100,000 Population.*

	1930	1931	1932	1933	1934	1935	1936	1937
Rate	35.7	34.4	32.7	28.6	24.7	26.5*	28.3*	27.2*
Relative Rate†	41.7	37.9	35.3	31.3	27.1	24.4	25.1	21.5

*Note effect of increased reporting concurrent with increased publicity.

†Based on a constant number of total reports which is the average of total reports for the eight years.

The rate for gonorrhea has declined continuously since 1931 (Table 4).

Table 4. *Reported Gonorrhea Expressed as Rate per 100,000 Population.*

YEAR	RATE	YEAR	RATE
1930	163.9	1934	150.7
1931	168.4	1935	142.0
1932	156.8	1936	139.1
1933	152.6	1937	133.0

In 1937, physicians reported 53.6 per cent of the gonorrhea cases, clinics 37.7 per cent and institutions 8.7 per cent; physicians reported 33.9 per cent of the syphilis cases, clinics 44.9 per cent and institutions 21.2 per cent. If institutions are disregarded and only those patients are considered who were free to choose between clinic and physician, the distribution of gonorrhea cases becomes 41 per cent and 59 per cent respectively, and that of syphilis cases 57 per cent and 43 per cent. The increasing number of reports of syphilis from physicians is probably due to improved reporting and better case-finding.

DISTRIBUTION OF CASES

The ratio of reported male to female gonorrhea and syphilis has shown very little variation since 1932. For the eight years 1930-1937, 1.4 times as many males as females were reported as having syphilis and 3.2 times as many males as females were reported as having gonorrhea. The relatively wide gap between reported male and female gonorrhea reflects the all-too-prevalent failure to diagnose gonorrhea in females, due perhaps to the facile acceptance of a negative smear as definitive proof of its absence.

The percentage of reported primary syphilis according to marital state in both men and women has been relatively constant since 1930 except for minor fluctuations (Table 5). The proportion of syphilis occurring in single men

has been consistently greater than half, the averages being 59.0 per cent in single men, 28.9 per cent in married men, and 7.2 per cent in those widowed, divorced or separated. The figures are virtually reversed for women: an average of 42.3 per cent in single women, 39.0 per cent in married women, and 13.7 per cent in those widowed, divorced or separated.

The figures for gonorrhea according to marital state parallel fairly closely those for syphilis: in men, 65.0 per cent of reported gonorrhea cases occurred in the single group, 26.9 per cent in the married and 4.7 per cent in the widowed, divorced or separated (Table 6). The corresponding figures for women are 48.6 per cent, 35.7 per cent and 11.9 per cent. The relatively high proportion of primary syphilis and gonorrhea in widowed, divorced or separated women may perhaps be ascribed to the carrying over of a number of infections actually contracted in the married state; otherwise it must be assumed that there is an increased rate of infection in that group.

Table 5. *Sex and Marital State in 3600 Cases (2836 Male, 764 Female) of Reported Primary Syphilis.*

Sex and Marital State	1930 %	1931 %	1932 %	1933 %	1934 %	1935 %	1936 %	1937 %	1930-37 Average %
Male:									
Single	64.8	60.5	60.6	62.7	56.3	58.9	52.8	56.1	59.0
Married	21.6	27.5	28.9	28.3	30.6	30.3	36.3	27.3	28.9
Widowed, divorced or separated	8.6	7.4	6.2	3.1	8.5	5.4	6.8	11.6	7.2
Not stated	5.0	4.6	4.3	5.9	4.6	5.4	4.1	5.0	4.9
Female:									
Single	44.9	52.1	37.6	35.5	43.3	49.0	40.0	35.6	42.3
Married	36.5	37.7	39.5	41.8	40.5	35.4	41.0	40.0	39.0
Widowed, divorced or separated	13.5	7.1	16.5	17.7	13.5	11.4	11.0	18.9	13.7
Not stated	5.1	3.1	6.4	5.0	2.7	4.2	8.0	5.5	5.0

Table 6. *Sex and Marital State in 52,188 Cases (39,320 Male, 12,868 Female) of Reported Gonorrhea.*

Sex and Marital State	1930 %	1931 %	1932 %	1933 %	1934 %	1935 %	1936 %	1937 %	1930-37 Average %
Male:									
Single	66.7	66.8	65.0	66.5	65.0	64.4	63.9	62.0	65.0
Married	26.0	26.6	26.9	26.2	27.0	27.8	26.3	28.6	26.9
Widowed, divorced or separated	3.8	3.8	5.2	4.3	4.6	4.2	5.4	6.0	4.7
Not stated	3.5	2.8	2.9	3.0	3.4	3.6	4.4	3.4	3.4
Female:									
Single	49.7	51.0	50.9	51.2	48.0	48.0	45.0	44.9	48.6
Married	36.3	35.5	34.8	34.4	36.3	35.5	35.9	36.8	35.7
Widowed, divorced or separated	11.2	10.1	11.0	11.3	11.7	11.9	13.2	14.8	11.9
Not stated	2.8	3.4	3.3	3.1	4.0	4.6	5.9	3.5	3.8

Whereas a much higher percentage of primary syphilis was diagnosed in males than in females, 12.5 per cent and 4.7 per cent respectively, the comparative percentages of early syphilis (including primary and secondary) were essentially the same, 27.9 per cent and 25.6 per cent respectively (Table 7).

The steadily decreasing percentage of reported early syphilis in both males and females since 1930 is a reflection partly of a declining reported incidence (Table 2) and to a greater degree of an increased reporting and therefore an increased percentage of late syphilis. In fact, so rapidly has the percentage of late syphilis increased that, in 1937, 75 per cent of reported female syphilis and 74 per cent of male syphilis was late syphilis, compared with 60 per cent and 54 per cent respectively in 1930. This marked increase is directly due to the commoner use of routine blood tests in hospitals and clinics and by physicians, with the consequent discovery of numerous cases of clinically unrecognized late syphilis. It may be expected that as this reservoir is depleted by discovery and reporting, the percentage of late cases will decline. The high percentage of late syphilis reported in any year indicates the need for education of the public so that the infected may seek medical attention early, and the need for education of the medical profession so that the infection may be recognized and adequate treatment instituted.

Table 7. *Percentage by Sex and Stage in 39,159 Reported Cases (22,600 Male, 16,559 Female) of Syphilis.*

Sex and Stage	1930	1931	1932	1933	1934	1935	1936	1937	Average
Male:									
Primary	16.7	14.9	14.4	13.7	11.8	9.5	10.8	10.4	12.5
Secondary	18.7	18.9	17.0	15.4	13.6	13.2	13.3	10.4	15.1
Total early	35.4	33.8	31.4	29.1	25.4	22.7	24.1	20.8	27.6
Late	60.0	60.2	64.0	64.6	69.0	71.9	71.9	74.7	67.2
Congenital	4.6	6.0	4.6	6.3	5.6	5.4	4.0	4.5	5.2
Neurosyphilis*	16.8	13.7	13.9	12.6	10.1	12.0	10.7	10.3	12.5
Female:									
Primary	6.7	5.4	5.6	4.2	4.0	4.3	4.2	3.3	4.7
Secondary	29.8	26.8	24.9	21.3	18.1	16.2	16.0	14.1	20.9
Total early	36.5	32.2	30.5	25.5	22.1	20.5	20.2	17.4	25.6
Late	53.7	58.1	59.6	66.0	70.2	70.6	73.0	75.2	65.8
Congenital	9.8	9.7	9.9	8.5	7.7	8.9	6.8	7.4	8.6
Neurosyphilis*	6.9	6.7	6.1	5.6	5.8	5.7	4.9	4.8	5.8

*Included in "late" percentage.

The percentage of congenital syphilis, including both males and females, has declined somewhat (7.0 per cent in 1930, 5.9 per cent in 1937). The con-

sistently higher number of female than male congenital syphilis cases reported is as interesting as it is unexpected, since the birth rate of males and females is approximately equal in this state, and it may be presumed that male and female infants have an equal opportunity to contract syphilis from an infected mother. The cause of this disparity is not clear.

It is to be hoped that the excessive proportion of congenital syphilis in both males and females will be reduced in the near future by the routine use of the blood test in pregnancy and the thorough treatment of every syphilitic pregnant woman throughout each pregnancy, regardless of the response of the blood test to treatment.

Table 8. *Percentage by Sex and Age in 52,188 Reported Cases (39,320 Male, 12,868 Female) of Gonorrhea.*

SEX AND AGE GROUP	1930	1931	1932	1933	1934	1935	1936	1937	AVERAGE
Male:									
0-14	1.0	0.7	0.8	0.6	0.7	0.5	0.8	0.7	0.7
15-19	8.9	8.3	7.8	8.1	7.2	8.3	7.2	6.0	7.7
20-24	32.7	31.9	31.8	31.0	30.4	29.7	29.6	30.3	30.9
25-29	24.4	23.9	25.2	24.8	26.0	24.3	25.7	24.9	25.0
30-34	14.4	14.6	14.2	15.7	14.8	15.6	14.2	15.1	14.8
35-39	8.5	9.9	9.4	9.2	9.5	9.2	9.8	9.0	9.3
40-49	7.2	7.9	7.6	7.8	8.5	8.9	9.4	9.5	8.4
50+	2.9	2.8	3.2	2.8	2.9	3.5	3.3	4.5	3.2
Female:									
0-14	11.8	12.5	9.8	10.8	9.0	11.5	12.5	11.1	11.1
15-19	18.9	19.8	19.5	18.3	18.1	16.3	13.6	14.6	17.5
20-24	29.6	29.1	28.9	29.7	29.8	27.8	28.8	28.7	29.0
25-29	18.4	16.5	17.7	17.2	17.7	19.1	16.7	18.9	17.7
30-34	9.9	10.3	10.0	9.9	11.7	8.5	12.4	10.8	10.4
35-39	4.8	6.3	6.3	5.5	5.8	7.0	6.1	7.2	6.1
40-49	4.7	4.2	5.3	5.1	5.7	7.0	6.8	6.4	5.7
50+	1.9	1.3	2.5	3.5	2.2	2.8	3.1	2.3	2.5

The percentage distributions of gonorrhea and early syphilis according to age group have remained fairly constant since 1930 (Tables 8 and 9). Both diseases predominate in the younger groups, the peak incidence of infections occurring earlier in females than in males. The high percentage distribution of female gonorrhea in the 0-14-year group is due principally to the reported cases of gonococcal vulvovaginitis. If this group is excluded and the percentage distribution of adult female gonorrhea is considered, the percentages for the groups above fourteen years become slightly (about 1.5 per cent) higher.

Table 9. *Percentage by Sex and Age in 10,300 Reported Cases (6186 Male, 4114 Female) of Early (Primary and Secondary) Syphilis.*

SEX AND AGE GROUP	1930	1931	1932	1933	1934	1935	1936	1937	AVERAGE
Male:									
0-14	0.7	0.3	0.7	0.4	0.3	0.1	0.0	0.0	0.3
15-19	6.3	4.4	2.2	3.3	2.3	3.5	3.2	4.2	3.8
20-24	26.2	23.6	22.0	22.0	17.8	19.8	18.3	19.6	21.2
25-29	24.2	22.3	20.8	25.7	24.3	22.0	23.1	21.1	22.9
30-34	14.2	16.7	19.4	15.9	21.8	15.0	15.5	14.5	16.6
35-39	11.6	14.0	13.2	13.9	13.8	14.1	14.0	11.7	13.3
40-49	10.8	13.8	14.4	12.5	14.8	15.1	16.1	15.5	14.1
50+	6.0	4.9	7.3	6.3	4.9	10.4	9.8	13.4	7.9
Female:									
0-14	1.4	0.8	2.0	0.8	1.5	0.9	0.2	0.0	0.9
15-19	14.5	14.0	15.0	13.3	8.2	7.3	8.2	8.5	11.1
20-24	32.1	32.9	24.1	27.7	29.9	27.6	29.5	32.8	29.6
25-29	20.1	19.7	21.4	22.2	24.4	25.5	20.8	20.8	21.9
30-34	11.3	14.6	17.4	14.2	16.6	13.3	14.8	12.3	14.3
35-39	8.7	7.8	7.9	8.9	9.1	9.7	10.7	9.6	9.0
40-49	8.6	7.4	9.0	10.0	7.6	10.6	10.9	10.8	9.5
50+	3.3	2.8	3.2	2.9	2.7	5.1	4.9	5.2	3.7

The migration of patients outside their communities of residence for treatment varies inversely with the size of the community and the availability of clinical facilities (Table 10). In general, the smaller the community the larger the migration. In communities of less than 20,000 population, over half the patients with gonorrhea and almost three fourths of those with syphilis sought treatment outside their own community. The total number of patients migrating for treatment for gonorrhea has steadily declined from 1543 in 1930 to 1176 in 1937, whereas patients seeking treatment for syphilis in communities outside their own have increased from 891 in 1930 to 1455 in 1937.

Table 10. *Migration of Patients with Syphilis and Gonorrhea from Community of Residence to Physician or Clinic Elsewhere.*

LOCALITY	MIGRATION FOR TREATMENT TO			TREATMENT LOCALLY BY			TOTAL NO. OF CASES
	PHYSICIAN	CLINIC	TOTAL	PHYSICIAN	CLINIC	TOTAL	
	%	%	%	%	%	%	
SYPHILIS							
Population of community:							
Less than 20,000	26.3	48.1	74.4	25.2	0.4	25.6	4,319
20,000-49,000	17.2	47.0	64.2	27.4	8.4	35.8	4,012
50,000-99,000	15.9	30.4	46.3	23.9	29.8	53.7	3,597
100,000 and over	6.4	14.6	21.0	24.4	54.6	79.0	7,299
Boston	1.14	0.08	1.2	20.3	78.5	98.8	12,997
Massachusetts	9.3	19.1	28.4	23.1	48.5	71.6	32,224*
GONORRHEA							
Population of community:							
Less than 20,000	34.4	20.6	55.0	44.9	0.1	45.0	7,338
20,000-49,000	24.1	21.1	45.2	46.0	8.8	54.8	6,541
50,000-99,000	20.0	15.6	35.6	42.3	22.1	64.4	5,598
100,000 and over	9.7	7.9	17.6	45.1	37.3	82.4	10,713
Boston	2.1	0.1	2.2	36.6	61.2	97.8	17,428
Massachusetts	13.9	9.7	23.6	41.8	34.6	76.4	47,618*

*Institutions not included.

The percentage of patients with syphilis migrating to physicians is much less than that of patients migrating to clinics, although there has been a distinct tendency in the last few years for clinic cases to fall and private physician cases to rise both in migrating cases and cases treated locally.

The reverse is true of patients with gonorrhea, since many more migrate to physicians than to clinics. The increasing number of patients not migrating have been absorbed about equally by local physicians and by clinics. Since clinics routinely report their cases of syphilis and gonorrhea, whereas physicians do so only occasionally, the proportion of patients, migrating and local,

treated by clinics is considerably lower than would appear from the figures.

Of patients treated locally it is interesting that a fixed proportion are treated by physicians regardless of the availability of clinic facilities. It is apparent that a considerable percentage of patients will seek private care whether or not clinics are available.

LAPSED CASES, SOURCES OF INFECTION AND CONTACTS

Since 1930, 26,371 cases have been reported by name. In 25,449 of these the patients discontinued treatment prematurely (50.9 per cent syphilis and 49.1 per cent gonorrhea); 908 cases were reported as sources of infection or contacts (31.5 per cent syphilis and 68.5 per cent gonorrhea), and 14 as occupational hazards. Physicians reported 5.7 per cent of the total gonorrheal cases and 9.3 per cent of the total syphilitic cases.

Cases of gonorrhea reported by name have declined from a peak of 1981 in 1933 to 1300 in 1937. In cases of syphilis, however, reports by name have increased from 1889 in 1934 to 2039 in 1937. It is hoped that a substantial reduction in the number of reports by name will be effected by the intense follow-up program now in practice in most of the clinics. There has been a definite upward trend in the percentage of cases of gonorrhea reported by physicians, from 5 per cent in 1933 to 14 per cent in 1937, whereas clinics have provided a decreasing percentage of reports with successive years, falling from 92 per cent in 1933 to 79 per cent in 1937. This is consistent with improvement in follow-up service by clinics.

Of the patients discontinuing treatment for gonorrhea, 90 per cent were males and 10 per cent females; of those reported as sources of infection and contacts, 24.4 per cent were males and 75.6 per cent were females. The respective figures for syphilis are 59.5 per cent males and 40.5 per cent females and 51.5 per cent males and 48.5 per cent females. There has been remarkably little variation in these percentages since 1930. It is apparent that men neglect treatment for gonorrhea much more frequently than do women, since nine times as many were reported by name as lapsing, whereas only three times as many men as women were reported as having gonorrhea.

The ratio of reported male and female sources of infection and contacts (1:3) is in inverse proportion to reported male and female incidence (3:1); this is to be expected since female sources of infection and contacts are discovered from men under medical care. The ratio of males and females reported for prematurely discontinuing antisyphilitic treatment (1.5:1) parallels closely the reported incidence of male and female syphilis (1.4:1), while the ratio according to sources of infection and contacts (1.1:1) approaches inversion.

The decline of total cases of gonorrhea reported by name has occurred in the lapsed group, where male cases have fallen from 1774 in 1933 to 1088 in 1937, and female cases from 171 in 1934 to 134 in 1937.

Of the patients reported by name and referred to local boards of health for follow-up, 47.9 per cent were found and returned to treatment (52.0 per cent of those with syphilis and 43.9 per cent of those with gonorrhea). A higher rate of successful follow-up is achieved by epidemiologists connected directly with the treating agency, reaching as high as 90 per cent success in some instances. This is due partly to the confidential relation between the patient and the worker, and partly to the fact that the cases followed represent the less refractory ones.

SUMMARY

An analysis of reports of syphilis and gonorrhea received by the Massachusetts Department of Public Health from 1930 to 1937 inclusive is presented.

During this period, 91,347 reports of syphilis and gonorrhea were received from 3028 physicians, 28 clinics and about 180 hospitals and institutions. Of the total reports, 39,159 represent syphilis in all stages, and 52,188 gonorrhea. The reports of total cases of gonorrhea have shown a steady decline since 1931, the cause of which is not clear. Increased reports of syphilis are due to improved reporting and increased routine blood-testing, and represent an increase in late cases. The rate per 100,000 population of early syphilis has declined since 1930, as has also the rate for gonorrhea. The respective distribution of reported gonorrhea and syphilis between clinic and physician was 41 per cent and 59 per cent for gonorrhea, and 57 per cent and 43 per cent for syphilis.

The ratio of male to female reported syphilis for the eight-year period was 1.4:1, and for gonorrhea 3.2:1. According to marital state, 59.0 per cent of reports of primary syphilis were in single men, 28.9 per cent in married men and 7.2 per cent in those widowed, divorced or separated. For women the figures were almost reversed, an average of 42.3 per cent of primary syphilis being reported in single women, 39.0 per cent in married women and 13.7 in those widowed, divorced or separated. The figures for gonorrhea were similar to those for syphilis. Sixty-five per cent of the reported gonorrhea cases occurred in the sin-

gle male group, 26.9 per cent in the married and 4.7 per cent in the widowed, divorced or separated. In women the corresponding figures are 48.6 per cent, 35.7 per cent and 11.9 per cent. The percentage of primary syphilis diagnosed in the male was much higher than in the female,—12.5 per cent and 4.7 per cent respectively,—although the percentage of male and female early syphilis reported was essentially the same, 27.9 per cent and 25.6 per cent.

The average percentage of reports of congenital syphilis was 5.2 for the male and 8.6 for the female.

The distribution of gonorrhea and early syphilis according to age group has remained fairly constant since 1930. Both diseases predominate in the younger age groups, the female being infected earlier than the male.

The proportion of patients migrating for treatment outside their communities of residence varied inversely with the size of the community and with the availability of clinical facilities. The total number of patients migrating for treatment for gonorrhea has steadily declined since 1930 (perhaps owing to a decline in total reports), whereas the reverse has been true of patients migrating for the treatment of syphilis (perhaps owing to an increase in total reports).

Since 1930, 26,371 cases have been reported by name. Of these, 25,449 were reported as discontinuing treatment prematurely, 908 as sources of infection or contacts, and 14 as constituting occupational hazards. Of the patients discontinuing treatment for gonorrhea, 90 per cent were male and 10 per cent female. Of those reported as sources of infection and contacts, 24.4 per cent were male and 75.6 per cent female. For syphilis the respective figures are 59.5 per cent male and 40.5 per cent female, and 51.5 per cent male and 48.5 per cent female.

About half the patients reported by name and referred to local boards of health for follow-up were found and returned to treatment.

545 State House.

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THE OPPORTUNITY FOR THE MODERN TREATMENT OF LOBAR PNEUMONIA IN GENERAL PRACTICE*

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THIS article is not a plea for the abandonment of the older, well-established methods of symptomatically treating lobar pneumonia. It aims rather to urge the more widespread adoption of the newer and more specific methods of treating this disease in general practice. The treatment to be outlined has been found to be entirely capable of application in a practice confined to a town of 1600 people, draining a surrounding country of about equal population. The methods can be used in the home as well as in the small country hospital. In the light of this experience it has been found that, within certain limits defined by economic considerations, the treatment of lobar pneumonia in such rural communities can and should approximate that available in the larger hospital centers. All that is necessary to bridge the apparent difficulties is the exercise of a little ingenuity which will often make the application of some of these newer methods surprisingly inexpensive.

In the home treatment of pneumonia the country doctor has to contend not only with the limited purses but with the unlimited prejudices of his patients. He must use the utmost tact in disposing of the importunate therapeutic suggestions or demands of overzealous friends and relatives of the patient. These range all the way from an onion rub to a skunk-oil poultice. Occasional concessions may have to be made, but by and large, the physician is wise to assert his authority at the outset and to "choose his own weapons." He must remember that under home treatment, the patient is more apt to be overtreated than undertreated. The patient should also be spared frequent, exhausting and unnecessary examinations, and the family should be reassured that physical signs take hours or days rather than seconds or minutes to develop.

NURSING CARE

It is generally recognized that good nursing care is one of the most important factors in the successful treatment of this disease. If possible, the physician should insist that a graduate nurse be employed. Here again he is apt to encounter strong prejudices; members of the family may assert, for example, that they "would rather wait

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on the patient than on the nurse." The family must be reassured that the present generation of graduate nurses is well able to take a hand in the general household, the condition of the patient permitting. It should also be emphasized that it is hardly logical to insist on a well-trained physician who is present for only a small part of the day, while entrusting the care of the patient during the greater part of the day to an untrained person. If the family cannot afford to pay a graduate nurse's fee, it will be necessary to employ a "practical" nurse. And in this case it devolves on the physician to teach the nurse to carry out the more important nursing procedures.

Serum Therapy

It has been definitely established that serum therapy diminishes the mortality in lobar pneumonia. This treatment saves a considerable number of patients who otherwise would die. Just as appendicitis is an acute surgical emergency, so is lobar pneumonia an acute medical emergency. It is not to be put off, for example, until "after office hours." An immediate etiologic diagnosis must be made. In a good proportion of cases the finding of the etiologic agent indicates what specific treatment is necessary. Since the physical signs of consolidation may not appear before the second or third day of the disease or not at all, and since serum therapy loses most of its effectiveness after the first few days, it must be started as soon as the type of the pneumococcus causing the pneumonia is determined (if, indeed, the type found is amenable to serum therapy). To label true cases of lobar pneumonia in which frank signs of consolidation have not yet appeared as "threatened pneumonias" and to wait for the development of signs, is to lose valuable time. In most cases the characteristic onset with chill, temperature over 102°F., cough, prostration, pain in the side, rapid respirations and bloody or rusty sputum is enough to make a clear-cut diagnosis of lobar pneumonia.

Once the diagnosis is made and the patient is comfortable, sputum should immediately be obtained and the type of pneumococcus, if any, should be determined. Often the patient has to be coaxed, or even taught how, before he is able to raise sputum. Depending on the accessibility of a bacteriological laboratory and the physician's ability to carry out the necessary serological procedures himself, the sputum will then either be sent to the nearest laboratory for typing or will be typed by the physician in his office. The new Neufeld technic has so cut down the time necessary to determine the type that only a few minutes are required. First a Gram stain should be done to determine whether a pneumococcus is present. The Neufeld technic denotes the type by the swelling of the capsule of the pneumococcus in the presence of specific (rabbit) antiserum. Tests should be carried out at least with Types 1 and 2, and where feasible, Types 3, 5, 7 and 8 should also be tried. The therapeutic value of Types 1 and 2 antiserums is quite well established, and favorable reports have appeared following treatment with Types 5, 7, 8 (now commercially available) and 14 pneumococcus antiserums.[2]

When no sputum is available it is important to try one of the other methods of identifying the organism rather than to wait for the sputum which may never be obtained. Pharyngeal swabbings may be cultured by dipping the swab into a sterile test tube containing 2 cc. of ascitic broth. The organism may be typed after four to six hours' incubation. The blood culture, another procedure that can be easily carried out in the home, may likewise enable the physician to identify the organism when sputum is not available. The commercially available tube, which sucks the blood from a vein directly into the culture medium, eliminates the elaborate technic of culturing blood used in large hospitals. The organism may be typed from the blood culture after twelve to twenty-four hours' incubation. Even when the pneumococcus has been identified from the sputum the blood culture may give valuable information. It keeps the physician informed as to whether bacteremia is present and may indicate the need for the continuation of serum therapy. It is also of great prognostic significance, persistently positive blood cultures being a bad omen.[3] In some cases a precipitin test on the urine may enable one to track down the offending organism. If the presence of a specific type of pneumococcus is detected in the urine, that type may be regarded as the etiologic agent, but failure to detect a specific type does not rule out that type as the etiologic agent for it is apparently only in the severe cases that a sufficient concentration of the "specific soluble substance" (pneumococcus polysaccharide) in the urine is attained to be capable of detection by this method. Diagnostic lung puncture or "lung suction" has recently been reintroduced for the early diagnosis and typing of the sputum-free case,[4] but until more is known about the risks attached to this procedure it would appear to be wise to refrain from using this method in general practice.

Since Types 1 and 2 pneumococci are rarely found in the throats of normal persons, the finding of either of these types may be regarded as

sufficient evidence that the organism is the etiologic agent in the particular case, and the specific antiserum should be given at once. Since Type 3 pneumococci and all those of the higher types are occasional inhabitants of the normal upper respiratory tract, it is wise, whenever organisms of these classifications are found, to repeat the typing on a subsequently obtained specimen.[5] The true etiologic agent may then be found to be a Type 1 or 2 pneumococcus and thus the opportunity to increase the patient's chances of survival will not be lost.

In severe weather when road conditions make traveling extremely difficult, if not impossible, there may be great delay in getting sputum typed. In such situations it is still considered justifiable to administer polyvalent (Types 1 and 2) antiserum without waiting for type determination. Beyond the waste of serum, the expense and the subsequent production of a sensitivity to horse serum, such a procedure does no harm and the possible benefits at the time of the emergency may more than outweigh these disadvantages. With the advent of the Neufeld technic, however, it seems that this "shotgun" method of treatment will not have to be used so frequently.

In children under twelve years of age several factors militate against the use of serum therapy. The death rate is low; the veins are difficult to enter, and repeated intravenous injections practically impossible; Types 1 and 2 pneumococci are found in a much lower percentage of cases than in adults; and no conclusive results are as yet available as to the value of serum therapy in childhood. Hence in the present state of our knowledge typing may be dispensed with in such cases. Nor should serum be given to elderly debilitated patients, moribund patients, patients with acute pulmonary edema or patients in stupor, coma or shock.

Before giving serum it must be determined whether the patient is sensitive to horse serum. If he has never been able to go near horses without developing asthma or hay fever, if he has had an injection of horse serum within the previous three months, if the conjunctival test with horse serum is positive, or if the intradermal test with diluted (1:100) horse serum is strongly positive, serum should not be given. Serum therapy may be very cautiously attempted in the absence of any of the above enumerated contraindications, in patients giving a personal or family history of allergic diseases, in those who have had horse serum injections more than three months previously or in those showing a mild intradermal reaction with diluted (1:100) horse serum. In cases falling in this group it is advised[6] that 0.5 or 1 cc. of the serum be first injected intramuscularly. If no untoward reaction develops within an hour, serum may be given intravenously by the usual method. If, on the other hand, a reaction does occur, desensitization must be carried out. This is a laborious and painstaking procedure but well worth the trouble and, fortunately, rarely necessary. To desensitize, the serum is injected subcutaneously at twenty-minute intervals, doubling the dose at each injection. The initial dose should be 0.01 cc., and after the 2.6 cc. dose, subsequent injections are given intravenously at half-hour intervals beginning with 0.1 cc. and doubling the dose at each injection. These intravenous injections must be given very slowly, and if any untoward reaction develops, the injection must be discontinued for a few hours then resumed at the last dose. After 5 cc. has been given the remainder of the serum may be slowly given at a single injection.

The danger of serum reactions is frequently offered as an excuse by those who do not believe in serum therapy. These reactions can now be largely avoided by observing the above precautions, by using the newer concentrated serums, by warming the serum to body temperature and by giving the initial injection very slowly (1.0 cc. per minute), stopping at the first sign of a shock-like, asthmatic or urticarial reaction or a chill. A small syringe containing 1.0 cc. of epinephrin (1:1000) should always be at hand, and this should be injected subcutaneously at once if any of these symptoms appear. In spite of this it must be admitted that our present methods of determining sensitivity are by no means perfect and, in very rare instances, unfavorable reactions cannot be avoided. Nor have we a way of preventing the late serum sickness, which, in a small percentage of cases, follows serum administration after a week or ten days. This occurs even with the newer concentrated serums. It is uncomfortable but entirely harmless. The use of antipneumococcus rabbit serum in patients sensitive to horse serum is now being investigated[7] and offers considerable promise.

As to the actual serum injection it must be pointed out that the commercially available syringe vials are quite impractical. The needles have long bevels, the rubber plungers are apt to stick during the injection, and the rate of injection cannot be well controlled. It has been found much more satisfactory to transfer the serum to an all-glass syringe provided with a short-beveled needle.

In general practice, particularly in a rural community, the accessibility of the patient's home necessarily determines the frequency of serum

administrations. When distances are great and road conditions are poor it is impractical, if not impossible, to give serum as frequently as is generally advised. A very workable plan is an initial injection of 20,000 units, a second dose of 40,000 units four hours later and a third injection of 40,000 to 60,000 units from four to six hours later. No more serum is given if a crisis has been produced in the meantime. If the temperature rises again or if the first or a subsequently obtained blood culture is positive, more serum is given.

The physician is often expected by the family to be present at the time of the crisis — for hours on end if necessary. However, the crisis is now regarded as an immunological event coinciding with the appearance in the blood of an optimal amount of the necessary antibodies. It has not been proved that anything which the physician does or does not do at the time of the crisis influences the attainment of this state. The unnecessarily melodramatic feeling among the laity that the doctor must be present to engineer the patient through this hazardous period has unfortunately been fostered at times by members of our profession.

The matter of the expense of serum therapy is one that cannot be glossed over lightly. Many families cannot afford to pay the current rate of twelve dollars per vial (20,000 units) for several vials. The doctor may emphasize, for example, that in human life equivalents, and dollar for dollar, the money is just as well spent for serum as for a surgical operation such as an appendectomy. But it is unfortunately one of the frailties of the lay mind to put more value on the surgeon's than on the medical man's technic. It will often be found wise and expedient to have the local druggist dispense the serum. He is usually a much better business man than the physician and much more apt to collect his bill for this costly item. When the serum is provided by the state, this worry is removed from the physician's shoulders. At any rate it is to be hoped that with the more widespread dissemination of knowledge concerning serum and its more extensive use the manufacturers will be able to sell it at a lower price.

Type 3 Pneumonia and Sulfanilamide

In the last few years numerous reports have appeared of the efficacy of sulfanilamide in combating certain infections, particularly those due to the streptococcus. In view of the biologic similarity of the highly virulent Type 3 pneumococcus (formerly called *Streptococcus mucosus*) to the streptococcus, it is not surprising that successes have been reported in treating Type 3 pneumonia with this drug.[8] Although it is as yet too early for instances of untoward reaction or sensitivity to have caught up with the early successes that attend any new treatment, it is difficult to believe here that the remedy is worse than the disease. Because Type 3 pneumonia carries a very high mortality rate and because there is no effective antiserum for it, other than rabbit serum, it would seem that we should keep our minds open as to the possibilities of this drug.

Oxygen Therapy

While there is no doubt regarding the effect of oxygen in alleviating dyspnea, delirium, distention, and so forth in many cases with marked anoxemia, there is still some controversy as to the precise indications for and the value of such therapy. Opinions range from the extreme of using oxygen routinely from the onset of all pneumonias to that of using it almost as a last resort.

Excepting the oxygen chamber, the most comfortable but unfortunately most expensive common method of administering oxygen is the use of the oxygen tent. The new open-top tent is relatively inexpensive. If none is available, one can be improvised by the use of an open-top box provided with a rubber attachment to fit snugly around the patient's neck. The oxygen enters the box, first passing through a small chamber filled with ice. The latter humidifies the oxygen and cools it so that it is less liable to rise out of the box. Or resort may be had to the nasal catheter method of oxygen administration.[9, 10] A very workable set-up consists of an oxygen tank equipped with pressure gauges, two one-gallon bottles, a glass Y tube, two nasal catheters and enough rubber tubing to allow the patient to turn easily from side to side. The gas is conducted from the tank to a bent glass tube reaching to the bottom of the first bottle, which is filled with water. The flow should be adjusted so that there is just short of a continuous stream of bubbles. This humidifies the oxygen and makes it less irritating. The gas is then led through the second bottle which is empty. This rids the oxygen of any excess moisture and makes it impossible for any free liquid to enter the patient's lungs. The oxygen is then led into the catheters which are attached to the two limbs of the glass Y-tube. Each catheter is first lubricated with glycerin or albolene and passed back into the nasal cavity until its tip is just visible behind the soft palate. It is then withdrawn about half an inch. The catheters are kept in position by attaching them to the cheeks with adhesive tape.

If oxygen is to be of value it must be given con-

tinuously. There is neither rhyme nor reason to the former practice of having the apparatus "on" for certain periods and "off" for others. When it is decided to discontinue oxygen it should, on theoretical grounds, be done gradually. Otherwise the abrupt change from a high oxygen tension (low altitude) to a low oxygen tension (high altitude) may produce a reaction resembling mountain sickness.

Speed[9] advises the use of oxygen for high temperature, rapid pulse and increased respiration (which must mean for practically all cases of lobar pneumonia), while Mathers's[11] more precise indications are cyanosis of the nail beds, a pulse rate over 120, a respiratory rate over 36 and the presence of delirium.

The face-mask funnel method of administering oxygen is inefficient. The recently introduced subcutaneous[12] and intravenous methods of giving it are still very much in the experimental stage and cannot be recommended in general practice.

At best, oxygen therapy can be regarded as only a symptomatic form of treatment. There is no conclusive evidence that oxygen saves lives; there is abundant evidence that serum does. Hence we cannot logically recommend oxygen for poor patients. If money can be obtained by pooling family resources, this money should be spent on serum or nursing care.

Treatment of Circulatory Failure

By and large the circulatory failure which occurs in pneumonia is the result of peripheral vascular collapse rather than of a primary heart failure, as was formerly supposed.[13] As a result of the anoxemia and the severe toxemia the walls of the smaller blood vessels — arterioles, capillaries and venules — lose tone, and the vessels dilate. This, in turn, leads to a pooling of the blood in the peripheral part of the vascular system. There is an inadequate venous return to the heart, and a shock-like condition supervenes. The stagnation of blood in the small vessels accounts in part for the intense heliotrope or cyanotic color of the skin and the failure of color to return promptly to an area which has been blanched by pressure. In accordance with the Bainbridge reflex,[14] the inadequate venous return is followed by an increase in the heart rate and a lowering of the blood pressure and of the pulse pressure. Other symptoms are extreme weakness and sweating. At first sight these signs and symptoms impress the observer as evidence of heart failure, but the rapid feeble heart is really the result of peripheral circulatory failure. By the same token treatment must be directed to the periphery of the circulatory system rather than to the heart. It aims to overcome the dilatation of the smaller blood vessels. It must be remembered that the atonic vessel wall, numbed by toxins and deprived of its normal quota of oxygen, responds to the therapeutic measures at hand much less readily than a normal vessel wall. Hence the results of treatment are often discouraging.

Oxygen therapy is claimed by some to be efficacious. Hydrotherapy, such as moderate cooling by an alcohol sponge, may produce vasoconstriction. "It is well known that local exposure of the skin to low temperature causes a definite paleness of the exposed skin and it can easily be verified microscopically that a contraction alike of capillaries, venules and arterioles is involved in this reaction."[15] Although the following drugs may be tried, it must be admitted that there is little evidence of their value. Epinephrin hydrochloride (1:1000) may be given subcutaneously in doses of 0.5 to 1 cc. every three or four hours or by continuous intravenous infusion in doses of 1 cc. to a liter of physiological salt solution or 5 per cent glucose. Ephedrine sulfate in doses of 3/8 to 3/4 gr. may be given by mouth or subcutaneously every three or four hours. Pituitrin in doses of 1 cc. every twelve hours has been recommended. This drug incidentally may have the additional beneficial action of helping to combat distention. Caffeine sodio-benzoate stimulates the vasomotor centers as well as the respiratory centers and hence may improve peripheral vascular tone by its central action. The dose is 7½ to 15 gr. subcutaneously, repeated every two hours if effective. The use of strychnine seems to be very extensive in this state. The dose ordinarily used, 1/60 gr. by mouth, is probably no more effective than a placebo tablet. The dose should be 1/20 to 1/10 gr. hypodermically, repeated every three to six hours.[16]

In the erroneous belief that primary heart failure is responsible for the symptoms, digitalis is often given. This may not only be unnecessary but actually injurious for it means another toxin with which the already intoxicated heart muscle has to deal. In fact there is evidence that in shock-like conditions (such as pneumonia), digitalis has the undesirable effect of diminishing the minute-volume output of the heart.[17] This is in line with the clinical studies of Niles and Wyckoff[18] which showed that the indiscriminate use of digitalis in lobar pneumonia consistently increased the mortality rate. The present procedure[18, 19, 20] is to give digitalis only for its usual indications — frank congestive heart failure and auricular fibrillation or flutter, conditions in which the drug would be given whether or not lobar pneumonia were present. If congestive

failure is present it is due not to the pneumonia but to antecedent or co-existent heart disease. When digitalis is given it should be given in full digitalizing doses. Because of the unfounded fear of rapid digitalization, it must be stated that full digitalization may be safely carried out in the course of twenty-four or thirty-six hours. There seems to be no particular virtue in the practice of holding digitalis in reserve or as a measure of last resort. If the indication exists for its administration, the drug should be given immediately.

Pneumothorax Therapy

Pneumothorax therapy has recently been reintroduced in the treatment of lobar pneumonia.[21] Its rationale, indications, method of administration and value are still too much a matter of controversy to warrant its use in general practice.

Summary and Conclusions

General practitioners, particularly those in rural communities, have been slow in adopting the newer advances in the treatment of lobar pneumonia. This is due in part to the natural lag in the avidity with which new things are taken up by physicians, to the inertia of tradition and to misinformation or ignorance, professional as well as lay. But it seems to be due more to the expense involved. The exercise of a little ingenuity in the improvisation of apparatus, however, can make the application of some of the newer methods surprisingly inexpensive. It is to be hoped that the more widespread use of serum will make commercial serum available at lower cost. The provision of serum by the state eliminates a burden of the patient but confers on the physician the obligation of its intelligent use.

Serum therapy, oxygen therapy and the rational treatment of circulatory failure are a few aspects of the modern treatment of lobar pneumonia capable of application in general practice.

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COLD AS A STANDARD STIMULUS OF BLOOD PRESSURE*

A Study of Normal and Hypertensive Subjects

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ONE of the outstanding characteristics of essential hypertension is the variability of the blood pressure. This is ordinarily noted, not only from visit to visit, but also from minute to minute.[1] The fluctuation is more marked in the earlier stages of the disease, when frequently one may find a drop of blood pressure to normal.[2] Under various stimuli, such as excitement, pain, cold, heat and exercise, the blood pressure of such patients varies markedly. However, the amount of fluctuation is variable, depending on the stimulus, its intensity and other factors. It has, therefore, occurred to various workers that if one could have a standard stimulus to apply to a patient, one might get a standard response of blood pressure in such a person and possibly be able to follow the effect of treatment and the progress of the disease. Furthermore, many normal young people are certain to develop essential hypertension some day. If it were possible to apply a standard stimulus to such persons, it might likewise be possible to determine a group in which an abnormally great reaction resulted from this stimulus. In other words, it might be possible to determine in advance the future subjects of essential hypertension.

This being assumed, much credit is due to Hines and Brown for their introduction of a standard stimulus, consisting of cold water applied to the hand. Their study was first reported in 1932, and has since been supplemented by them in other papers. Since 1932 the test has been used by various workers, but unfortunately the technic of Hines and Brown has not been followed exactly. The present study is the first repetition of their study with their exact technic in a group of normal and hypertensive subjects.

LITERATURE

It is well known that the vasomotor center can be stimulated by many factors, such as lowered oxygen intake, increase of hydrogen-ion concentration in the blood either by carbon dioxide or other metabolites, stimulation of sensory nerves as by cold, pain and electricity, and emotional disturbances. The use of some of these stimuli to raise blood pressure and to study the vasomotor reactions in human subjects has long been carried out. Thus, Hertzell[3] in 1913 advised a test for arteriosclerosis in which the circulation to three extremities was arrested and the blood pressure was measured in the remaining extremity. He found that if the vessels were healthy there was but little rise in the other arm, while in cases of sclerosis the blood pressure rose to 60 mm. Similarly, the effect of a piece of ice placed on the hand was studied by plethysmograph, to determine whether vasoconstriction would occur, by von Romberg in 1904, O. Müller in 1906 and others.[3] Baráth[4] studied the effect of measured exercise on the blood pressure of normal subjects and individuals with hypertension and with arteriosclerosis. Müller[5] observed the influence of baths on blood pressure. Raab[6] studied the effect of carbon-dioxide inhalation and found that patients with hypertension had twice as great an elevation in blood pressure as had persons with a normal pressure. However, none of these methods have been satisfactory for clinical application, owing to their lack of simplicity.

Many studies have shown that there is a distinct tendency of the disease to be hereditary. One of us (D. A.)[7] has shown that there is a very high incidence of elevated blood pressures in the children of parents with essential hypertension. To be sure, these blood pressures might go up only momentarily; nevertheless they rise to levels differing from the slight rise in children of parents with normal blood pressure. It was stated in that study that this reaction in children was probably due to the excitement of the examination. In other words, the excitement caused by taking the blood pressure of children in hypertensive families was a form of standard stimulus which in children of normal parents caused no rise in pressure. It is clear, however, that such an emotional stimulus is variable and might not be effective in many people.

The advantage of the method devised by Hines and Brown[8] is that it can be used in a practical manner. They simplified the use of cold so that it could be applied in standard fashion to the hand, and the effect upon blood pressure could be noted. Their technic has been followed in the present study. The test consists essentially of plac-

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ing the hand in cold water up to the wrist; the temperature of the water should always be between 4 and 5°C., and as near 4°C. as possible. After the proper basal level of blood pressure has been obtained on rest, the hand is placed in the water and the blood pressure is measured in the other arm at the end of thirty seconds and of sixty seconds. The hand is then removed from the water and the blood pressure is determined at frequent intervals for a few more minutes until it has returned to its original level. In 1936, Hines and Brown[9] reported results of the test in 571 normal and hypertensive subjects. They were able to separate empirically a group of 288 normal people, in whom the mean rise of systolic blood pressure was 11.4 mm. of mercury, and of diastolic blood pressure 10.6 mm. They set an upper limit to normal rise of 22 mm. and termed those who fell in this group "hyporeactors." They found a group of 90 people who had normal blood pressure before the test, but who with the test had a mean rise of 29.4 mm. systolic and 24.5 mm. diastolic, and all of whom had a rise greater than 22 mm.; these were called "hyper-reactors." On the other hand, they found that with 127 patients with "organic" hypertension (with changes in the fundi), the mean rise was 47.2 mm. systolic and 34.3 mm. diastolic. The "preorganic" group of 66 hypertensive individuals (without changes in the fundi) had a mean rise of 34.4 mm. systolic and 25.4 mm. diastolic. In other words, the rise was from two to four times as great in the hypertensive group as in the normal one. Hines and Brown believed that the subjects who had a normal blood pressure with an increased reaction were probably members of hypertensive families, and might be candidates for the disease in the future. In support of this theory, they found that 84.6 per cent of the hyper-reactors and 86.6 per cent of the hypertensive subjects had a positive family history of cardiovascular disease, as compared with 17.2 per cent of the hyporeactors. Further evidence that hyper-reactions are of significance is contained in a recent paper[10] in which Hines reported the cold responses of 256 members of 30 families. He found that when both parents had normal blood pressures and gave normal responses to the cold test, all the children were normal reactors, but that when both parents had essential hypertension or had normal blood pressures with hyperactive responses, 95.0 per cent of the children had hypertension or were hyper-reactors. If one parent was a normal reactor and the other had hypertension or a hyper-reactive response, 43.4 per cent of the children had hypertension or hyper-reactive responses. These results parallel our own findings using no stimulus other than the emotional one of taking the blood pressure.[7] Finally, Hines and Brown have seen several hyper-reactors develop hypertension in the course of later observations.

As to the mechanism of the reaction, a tourniquet applied to the arm does not prevent the response, this fact ruling out the local production of a chemical or hormone as the pressor agent. The argument of Hines and Brown that the response is too rapid to be due to hormone produced elsewhere is not adequate. However, response occurs in Addison's disease and in completely adrenalectomized dogs, and this rules out adrenine as a major factor. The mechanism of the reaction is probably due to stimulation of the nerves of the hand, although Hines and Brown reached this conclusion by elimination. To prove this theory, the test should be carried out on an extremity completely devoid of sensation to cold and pain, such as is found in syringomyelia.

In 1933, Briggs and Oerting[11] repeated this work and studied 130 normal and hypertensive persons. They used an average temperature of 4.3° C. and kept the hand in the water for three minutes. Despite this deviation from the standard method of Hines and Brown, which requires the hand to be held in water only one minute, the response of blood pressure was very much less. The average systolic rise in normal subjects was 9.7 mm. and the diastolic rise 9.3 mm. In hypertensive patients the average systolic rise was only 16.4 mm. and the average diastolic rise 7.6 mm. Sedatives were omitted before the test. On the other hand, in 8 luetic patients, who had tabes but no cardiovascular lues, except for 1 case of aortitis, and all of whom had a normal blood pressure at the start of the test, there was an average rise of 49 mm. systolic, and 25 mm. diastolic. Data from a later group of luetics[12] led to the conclusion that the first series happened to consist of prehypertensive individuals. The investigators also studied a few cases of heart failure with normal blood pressure, and having obtained a marked response in 1 patient concluded that this indicated previous hypertension. They found that this test caused much discomfort, especially in patients with hypertension, many of whom cried as a result of it.

In 1935, Dieckmann and Michel[13] carried out the cold-pressor test in 190 pregnant and nonpregnant women, but used a temperature of 1° C. and an exposure of two minutes. In 5 patients with chronic nephritis they obtained severe reactions. In 18 normal pregnant women without a family history of essential hypertension the aver-

age systolic rise was 19.5 mm. and the average diastolic rise 19.8 mm. In 42 normal non-pregnant women without such a history the average systolic rise was 18 mm. and the diastolic rise 23 mm. In 28 normal non-pregnant women with such a history the average systolic rise was 35 mm. and the diastolic rise 34 mm. In 13 normal pregnant women with such a history the average systolic rise was 35 mm. and the average diastolic rise 26 mm. In 33 pregnant women with chronic nephritis the average systolic rise was 51 mm. and the average diastolic rise 37 mm. When the tests were repeated postpartum the reactions remained unchanged in all these groups. In 6 patients with symptoms of vasomotor instability but no family history of essential hypertension the average systolic rise was 37 mm. and the average diastolic rise 35 mm. In 6 patients with essential hypertension the average systolic rise was 36 mm. and the average diastolic rise 36 mm. They offered evidence that the cold response was a pain reflex; for between labor pains it was equal to the systolic rise in blood pressure recorded during the labor pain without any cold application. In a paper now in press, Dieckmann, Michel and Woodruff[14] have reported the results of the test in over 400 pregnant women. Of these, 152 patients who were normal at the time of the test and who did not deliver for at least three months were used in their analysis. Sixty-two patients had a systolic response of 29 mm. or less and of these only 2 developed toxemia, while 5 others had transient signs. Ninety patients had systolic responses of 30 mm. or more; of these hyper-reactors 15 developed toxemia and 15 others had abnormal vascular-renal symptoms. Dieckmann, Michel and Woodruff decided that a past history of toxemia or a borderline systolic blood pressure of 130 to 140 mm., together with a hyper-reactive response, was adequate evidence to justify advising the patient not to become pregnant.

In 1934, Randall, Murray and Mussey[15] reported the use of this test in pregnancy. They used the technic of Hines and Brown in every detail. They concluded that by means of the test it might be possible to pick out during early pregnancy a group of women some of whom might later develop hypertensive toxemia. They have not since then published any further data indicating whether the normal reactors among the pregnant women would or would not develop toxemia. However, they have recently concluded that the hyper-reactors are very liable to develop toxemia; they encountered some patients who seemed to react to the cold test with vascular collapse, and observed syncope following the test.[16] In 1937, Briggs and Oerting[17] reported another study of the test, this time in 233 unselected pregnant women, having presumably used the same modified technic as before. They found no case of hypertensive toxemia in the normal reactor group, whereas cases often occurred in the hyper-reactor group. They did not publish the average responses of their groups.

In 1936, Pickering and Kissin[18] concluded that a relatively large rise of blood pressure in response to a cold stimulus was not peculiar to potential or developed cases of essential hypertension. They studied only 32 patients. In 9 subjects with normal blood pressure and an average age of fifty-three, the cold stimulus caused an average rise of 22 mm. systolic and 11 mm. diastolic. In 12 patients with essential hypertension and an average age of fifty-four, the test caused a similar average rise of 21 mm. systolic and 13 mm. diastolic. These authors stated that the rise in blood pressure produced by the cold test is "fairly constant for each individual, that it tends to be larger in old subjects than in young, and that its size seems to be related to the degree of discomfort which the subject experiences, but is to a large extent independent of the resting blood pressure." Pickering[19] has stated that all the patients studied were ward inmates who had been in bed for days or weeks when the tests were done. Since many patients who have essential hypertension when ambulatory have normal blood pressure when confined to bed, many of Pickering's normal subjects may have actually had hypertension. Further, he also wrote that the blood pressure was measured for ten minutes or more before the test. Such comparatively brief periods prevent an adequate drop of blood pressure to a basal level, and would result in only a slight rise of pressure in response to the cold test. Also, the authors did not follow the technic of Hines and Brown exactly; for although they used a temperature of 4°C., they exposed the hand for three minutes instead of the standard one minute.

Finally, Yates and Wood[20] carried out the cold test in 205 subjects, in whom the blood pressure was less than 140 systolic and 90 diastolic. Although the ages were from eight to seventy, only 34 were more than forty. Analyzing the responses to the cold test at different age periods, Yates and Wood found no appreciable decrease in the percentage of abnormal responses in older age groups. They suggested that if abnormal response in early life means that essential hypertension will develop later in life, one would expect that at midlife the group of persons who still have normal blood pressure would have a small

incidence of abnormal cold responses. However, they studied only 13 subjects of fifty or older.

It is to be noted, therefore, that no study has as yet repeated the exact technic of Hines and Brown in essential hypertension.

TECHNIC OF TEST

The patients were seated in a quiet room, except for a few who were recumbent in bed on the ward, and the blood pressures were taken over variable periods until they remained at a constant level. This rest period varied between twenty and forty minutes, and usually four to five readings were made. In the majority of patients, repeated previous observations, carried out by having the patient rest for twenty minutes, during which time blood-pressure readings were made, had given us the fluctuations of the blood pressure. In the present study the blood-pressure cuff was placed on the arm resting on a table, while the opposite arm rested near a basin of ice water at a temperature of 4°C. When the blood pressure had fallen to its lowest level the free hand was placed in the water up to the wrist. The pressure readings were made at the end of thirty seconds and of sixty seconds, with the hand in the water during the entire interval. In this way about seventy seconds with the hand in the water elapsed before the second reading was completed, as carried out by Hines.[21] Both readings were recorded. The hand was then removed from the water, and the blood pressure was taken at once and a few minutes later.

RESULTS OF TEST

The tests were carried out in 48 subjects with normal blood pressure and in 88 subjects with hypertension, chiefly of the essential variety. The average age of both the normal subjects and of those with hypertension was fifty. The age range of the former was fourteen to seventy-six, and that of the latter twenty-four to sixty-eight. There were 21 men and 24 women in the normal group, and 18 men and 56 women in the hypertensive group.

In some of these 136 subjects the test was repeated many times, and in most of them twice, so that a total of 313 tests were carried out. The tests were often repeated on separate visits to the clinic. In all subjects, and on practically every test, we made certain that no sedative had been taken within a few days.

We arbitrarily divided our results into the same groups as did Hines and Brown.[8] In Group 1, composed of subjects with normal blood pressure, Hines and Brown placed those who reacted with a rise in systolic or diastolic blood pressure between 0 and 22 mm., the latter being the upper limit of normal. In our Group 1* there were 38 subjects. The average pressure increase of this group was only 10.5 mm. systolic and 9.0 mm. diastolic. From this low mean rise it is seen that the majority of this group were well below 22 mm. of rise, and most of them below 15 mm. Ninety-eight per cent of the group had a peak of less than 145 mm. systolic, and 92 per cent had a ceiling of less than 95 mm. diastolic.

Group 2, called normal hyper-reactors, consisted of 10 subjects in whom the blood pressure was normal. However, in these subjects there was an average rise of 33.0 mm. systolic and 17.8 mm. diastolic following the cold-pressor test. The average age was fifty-nine. We have not followed this small group long enough to discover whether any subjects have developed true essential hypertension. Sixty per cent of the group had a ceiling value of more than 145 mm. systolic, while 30 per cent had a diastolic pressure rise to above 95 mm.

In Group 3 we had 88 hypertensive subjects whose ages varied from twenty-four to sixty-eight, and in whom all degrees of the disease existed. We did not analyze their reactions according to degree of hypertension or secondary vascular changes. The average rise of blood pressure in this group, following the cold-pressor test, was 33.5 mm. systolic and 19.0 mm. diastolic. This rise was computed using the highest response to the test in each case if more than one test was carried out. If, on the other hand, we used the lowest range following the test, and averaged these results for the entire group, there was an average rise of only 23.1 mm. systolic and 16.0 mm. diastolic. We also noted that the return of the blood pressure to the previous basal level following the test was slightly slower than was the case in the normal group. However, in the vast majority of the patients the blood pressure immediately following the test dropped close to the basal level, but complete return to that level did not occur for some time. In all subjects of the hypertensive group the blood pressure was at an abnormal level to start with, so that we naturally obtained abnormal ceiling values. The variation in different tests, which resulted in the average rise for the group being much higher when the highest tests were used than when the lowest results were used, is due to the fact that it was not always possible to secure the same basal level before the test at different visits.

DISCUSSION

Measured cold caused some rise in blood pressure in 98 per cent of all the subjects tested. The test caused a rise in blood pressure two to four

*Normal blood pressure was considered to be less than 140 systolic, 90 diastolic, whereas a pressure greater than 160 systolic, 100 diastolic, was judged to be evidence of hypertension.

times greater in the hypertensive patients than in the subjects with normal blood pressure.

In certain respects our results differ from those of Hines and Brown. The latter found that the response to the test in an individual was remarkably constant from day to day, not varying more than 10 per cent. This constancy we have not experienced. We have obtained variations of response up to 100 per cent on different visits, chiefly in hypertensive patients, despite starting the tests in such patients at the same level at each visit. This has been true of both systolic and diastolic levels, although more frequent and marked in the former. Such variations have occurred in tests repeated within one hour. Similarly, in a given patient, tests repeated at the same or at separate visits with a different basal level at each test did not necessarily result in a similar ceiling level's being attained. In several patients, repeated tests were made over a period of many months and no correlation was found between the range of the blood-pressure response and the basal level at the start of the test.

In most cases, however, the amount of response depended on the basal level obtained before the test: the lower the basal level, the greater the response. This was true especially in the hypertensive patients, but also to some extent in the subjects with normal blood pressure. We also noted that mild doses of sedatives such as phenobarbital, in doses of ¼ gr. four times a day for one month, did not abolish the reaction. Rather, in some cases the reactions were just as great, or coincidentally even greater response was obtained. However, sedation to the point of marked drowsiness and general anesthesia, abolished the reaction to the cold test.

It was also observed that in a high percentage of patients the ceiling level of response to the cold test was close to the blood-pressure reading made at the beginning of the rest period before the test. In other words, the first reading of the blood pressure on entering the clinic, before any rest, was an excitement reading, a vasomotor response to emotion, and so forth. Unless one takes the blood pressure as soon as the subject is seated, this high reading will often be missed. When we allowed the patient to rest, so that his blood pressure dropped to a basal level, the application of the cold stimulus acted to raise the blood pressure to or near the original level at the start of the rest period. In many cases, however, the cold test did not cause a response or ceiling equal to the original blood-pressure reading at the start of the rest period, before the test. On the other hand, the response to the cold test often exceeded the highest blood pressure recorded during the rest period. Finally, it was noted that in patients who had been seen over a long period of time in the clinic, the cold test only occasionally gave a reading higher than the highest obtained at some time or other in the clinic.

Two mild reactions were encountered. In 1 hypertensive patient, nausea occurred during and immediately after each of two tests without any abnormal blood-pressure reaction. In another patient, a man with normal blood pressure, weakness and faintness occurred during a test, with a drop of blood pressure from 140 systolic, 90 diastolic, to 90 systolic, 70 diastolic, returning to normal in fifteen minutes, and without other apparent ill effects.

The final significance of the excessive reactions in certain people with normal blood pressure will be known only after these subjects have been followed for a number of years in order to note how many develop true essential hypertension. It does not seem likely that 95 per cent of the children of parents both of whom show essential hypertension or hyper-reactive responses to the cold test will develop true essential hypertension, despite the finding this percentage reacted abnormally to the cold test. For the present, therefore, the value of the cold test appears to lie in the field of investigation rather than in that of practical application. It is possible, however, that it will eventually prove of real value in general practice to pick out many future candidates for essential hypertension or hypertension during pregnancy or those with past hypertension.

From the investigative aspect, the test is simple to carry out, but from the practical point of view it requires the availability of ice and a thermometer and the maintenance of constant temperature. We have experimented with other pressor stimuli and have found a simple effective pressor stimulus — breath holding — requiring no apparatus. This test will be reported shortly.

SUMMARY

The literature describing the use of the cold-pressor test of Hines and Brown has been critically reviewed. No study was found that had repeated the same technic as Hines and Brown in a group of patients with essential hypertension. The present study, using the exact technic of Hines and Brown, has been repeated in 48 subjects with normal blood pressure and 88 subjects with essential hypertension. The results were in close agreement with those of Hines and Brown.

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HUMAN AUTONOMIC PHARMACOLOGY*

XIX. The Effect of Mecholyl, Prostigmin, Benzedrine Sulfate and Atropine on the Urinary Tract: Urographic Studies

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BOSTON

IN another communication[1] we reported the effects of Mecholyl and Prostigmin on the bladder as revealed by cystography. Because of the possible mechanical interference with the physiology of the urinary tract, which is inherent in this method, intravenous urography was utilized in the present study, in which Mecholyl (acetyl-beta-methylcholine chloride), Prostigmin (dimethylcarbamic ester of m-oxyphenyl-trimethylammonium methyl sulfate) and also Benzedrine Sulfate (benzyl-methyl carbamine) and atropine were administered.

MATERIAL AND TECHNIC

Control intravenous urographic studies were made on 9 passive co-operative patients with dementia praecox. Three of these were eliminated from the investigation because the x-rays showed various abnormalities. At intervals of three days to one week each subject received: (1) Mecholyl; (2) Mecholyl and Prostigmin; (3) Benzedrine Sulfate; and (4) Benzedrine Sulfate and atropine. In 2 of the cases Prostigmin alone and in 2 other cases atropine alone were administered. The time between successive plates was the same or approximately the same in the control studies and in those in which the drugs were given. No attempt to regulate the fluid intake was made, but it is known that the subjects drank practically only at mealtimes.

RESULTS

Effect of Mecholyl

Mecholyl (20 mg.) was administered subcutaneously immediately following a Diodrast injection, or the Diodrast was given at the height of the Mecholyl reaction (sweating, lacrimation, salivation, fall in blood pressure and increase in pulse rate), which occurs within ten minutes after the injection of the drug. Three or four plates were taken at intervals of five to ten minutes. A fifth and sometimes a sixth plate were taken at intervals of fifteen to thirty minutes.

Definite changes were visualized throughout the entire urinary tract in all 6 cases. The kidneys showed a decrease in size as much as 3 to 4 cm., in both length and width, as compared with the control plates. The pelves showed contraction as much as 3 to 4 cm. The calyces were narrowed and sharply outlined. The ureters became narrowed and sharply defined, and when visualized, showed evidence of increased peristalsis. The bladder was much smaller, triangular in shape with flattened top, markedly trabeculated and bolder in outline than in the control plates; it showed evidence of contraction for more than one hour after the injection of the drug. (Figures 1, 2 and 3.)

Effect of Mecholyl and Prostigmin

These two drugs were administered as follows: 0.5 mg. of Prostigmin was injected subcutaneous-

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ly, followed fifteen minutes later by the subcutaneous injection of 5 mg. of Mecholyl. They were used in these small doses because we have observed in previous experiments on their synergism that larger amounts are hazardous. In the above-designated doses, the effects are liable to be more marked than those produced by 25 mg. of Mecholyl alone. At the height of the combined response to the two drugs, that is, usually within five minutes after the administration of Mecholyl,

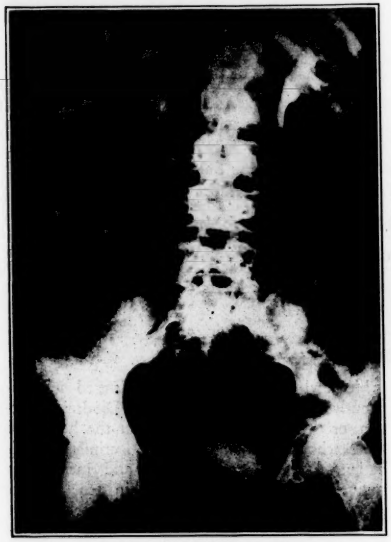

Figure 1. *Control Plate: Fifty-one minutes after the injection of Diodrast. The bladder is somewhat small but has a rounded shape. All subsequent illustrations are reproductions of urograms of this same subject.*

the Diodrast was injected. Plates were then taken as in the previous experiments with Mecholyl alone.

In 3 of the cases similar but more marked changes and in the other 3 cases similar but less marked changes occurred than those following Mecholyl alone in the kidneys, pelves, ureters and bladder. The more marked the general systemic reactions, usually the greater the changes visualized in the urinary tract. (Figure 4.) In 2 cases 0.5 mg. of Prostigmin alone revealed no visible changes in the kidneys, ureters or bladder.

Effect of Benzedrine Sulfate

Each subject was given Benzedrine Sulfate (20 mg.) by intravenous injection. By this route the blood pressure usually reaches its maximum within five minutes and gradually falls over a variable period, not reaching its original level in some cases for more than one hour. The Diodrast was given ten to twenty minutes after the injection of Benzedrine Sulfate. Five minutes later the first plate was taken. Four or five successive plates were taken at intervals of seven to ten minutes.

The following changes in the films were visualized in all 6 cases. In the kidneys there was penetration of the dye throughout so that they stood out much more boldly than those in the control or the Mecholyl plates. The kidneys were definitely larger than those in the control plates,

Figure 2. *The Effect of Mecholyl (20 mg. subcutaneously): Twenty minutes after injection. The calyces are contracted; the bladder shows marked contraction.*

the increase usually measuring 3 to 4 cm. in both diameters. The pelves and calyces were definitely larger but not so sharply visualized as in the control plates. The ureters were larger and more relaxed in appearance as compared with the controls. The bladder was usually enlarged to more than twice its original capacity; the dye was present in much less concentration, so that the organ had the appearance of a cord bladder. (Figures 5 and 6.)

Effect of Benzedrine Sulfate and Atropine

The 6 subjects were given 20 mg. of Benzedrine Sulfate and 0.6 to 0.9 mg. of atropine, both intravenously, and the technic used with Benzedrine Sulfate alone was followed.

The kidneys, although more visible than in the control plates, were much less plainly seen than those observed following the giving of Benzedrine Sulfate alone. The pelves and ureters had a vague diffuse appearance. The bladder was much enlarged, sometimes more markedly so than that seen following Benzedrine Sulfate alone. (Figure 7.)

Figure 3. *The Effect of Mecholyl: Thirty minutes after injection. The pelves and calyces are contracted; the bladder is very contracted, triangular in shape, with a flattened top, and markedly trabeculated.*

Two of the 6 subjects were given 1.2 mg. of atropine intravenously. The pelves, calyces, ureters and bladder all showed definite dilatation. The kidneys were more visible than those in the control plates but definitely less so than those taken after Benzedrine Sulfate. (Figure 8.)

DISCUSSION

The above-recorded observations on the changes in the size and tone of the pelves, ureters and bladder were not unexpected, since they are similar to those seen in the gastrointestinal tract following the administration of Mecholyl and Benzedrine Sulfate.[2, 3] On other autonomic functions, with a few exceptions, the action of these drugs corresponds to stimulation of the parasympathetic and sympathetic systems respectively.[4] In the urinary tract, however, with the exception of the bladder it is difficult to draw such an analogy, largely because the neurology of the kidney and ureter is still vague. Stimulation of the splanchnic nerve (sympathetic), for example, results in constriction of the renal vessels, an effect which does not correspond to the action of Benzedrine Sulfate, which produces dilatation of the renal vessels, if this be the correct interpretation of the enlargement and the increase in visualization of the kidney following the administration of the drug. Nor can the action of Mecholyl, which results in diminution in the size of the kidney, probably as the result of renal vasoconstriction, be compared to stimulation of the vagus, for this nerve contains no known vasomotor supply to this organ. As with other autonomic functions, however, Mecholyl and Benzedrine Sulfate probably act directly on the muscle of the renal ves-

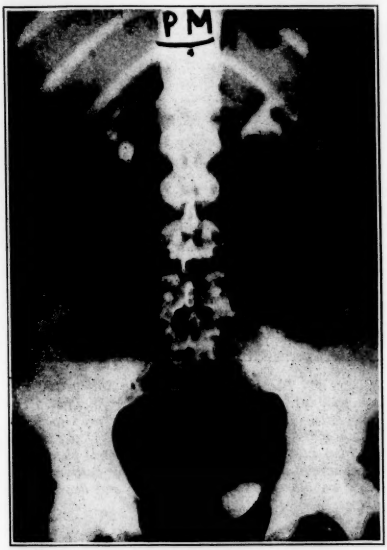

Figure 4. *The Effect of Prostigmin (0.5 mg. subcutaneously) and Mecholyl (5 mg. subcutaneously): Thirty-three minutes after injection. There is marked contraction of the pelves, calyces and ureter with increased peristalsis of the latter organ; the bladder shows marked contraction.*

sels, in the former case causing a vasoconstriction, and in the latter a vasodilatation.

We are able to find little data concerning the action of the autonomic drugs on the ureter. Elliott[5] was able to observe no reaction of the musculature of the ureter to stimulation of the

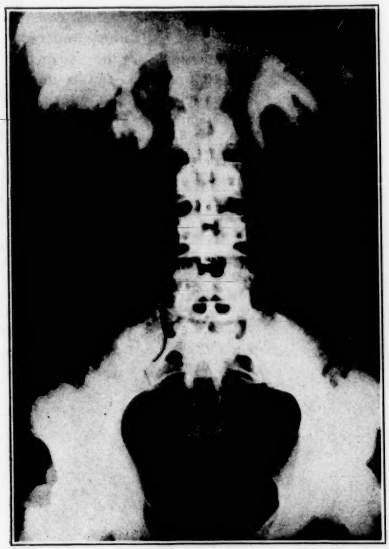

Figure 5. *The Effect of Benzedrine Sulfate (20 mg. intravenously): Thirty-five minutes after injection. The kidneys stand out sharply and are larger by 3 to 4 cm. in both width and length than in the control plate. The pelves are 3 to 4 cm. wider than in the control plate. The calyces are not sharply defined. The lower portions of the ureters are dilated; the bladder is large and hazy in outline.*

hypogastric nerve (sympathetic) or to the administration of adrenalin. Samaan and his collaborators[6] showed that syntropan, enatin, bromsalizol and eupaverine, which may be considered to have a mixed, although predominantly an adrenergic, action, all relax strips of the human ureter when in a state of spasm. Kinoshita[7] showed that a cholinergic group of drugs produced transitory spasm and increase of force of the urinary flow, and stimulated the tone of the ureter. However, there are at present insufficient data to allow one to compare Benzedrine Sulfate with stimulation of the sympathetic nervous system, or Mecholyl with stimulation of the parasympathetic nervous system. Here, again, these drugs probably operate independently of the autonomic nervous system, acting directly on the entire musculature of the pelves, calyces and ureter — Mecholyl causing contraction and Benzedrine Sulfate relaxation of these tissues.

In contrast to the kidney and ureter, the action of Benzedrine Sulfate and Mecholyl on the bladder corresponds to stimulation of the hypogastric nerve (sympathetic) and the pelvic nerves (parasympathetic) respectively, resulting in the former case in relaxation of the detrusor muscle, and in the latter case in increased tonus of this muscle. On human male subjects under spinal anesthesia, Learmonth[8] found that intravenous injections of adrenalin in appropriate dosage caused an immediate active dilatation of the bladder,

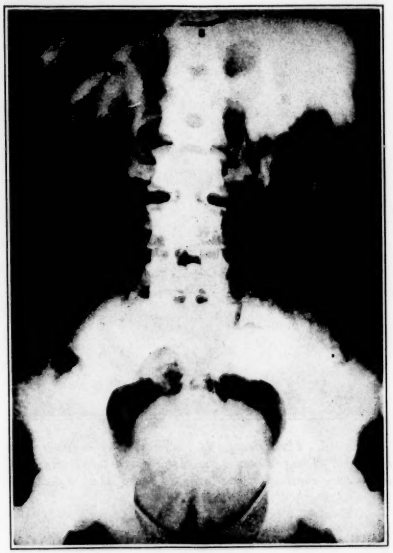

Figure 6. *The Effect of Benzedrine Sulfate: Fifty-five minutes after injection. The kidneys are still plainly visualized; the pelves, calyces and ureters are still markedly dilated. The bladder is greatly enlarged and has the appearance of a cord bladder.*

which remained at its increased capacity for approximately five minutes. No comparable experiments with cholinergic drugs, except those in the present study, are known to us. Cystometric studies and the stimulation of micturition produced by Mecholyl indicate that this drug increases the tone of the detrusor muscle of the

bladder. It must be stressed here that studies involving mechanical procedures, such as retrograde pyelography and cystography, are apt to interfere with the pure action of a drug on the urinary organs. This is indicated by our previous study[1] in which no definite changes in the bladder following the administration of Mecholyl were observed. This objection is obviated by intravenous methods of visualizing the urinary tract.

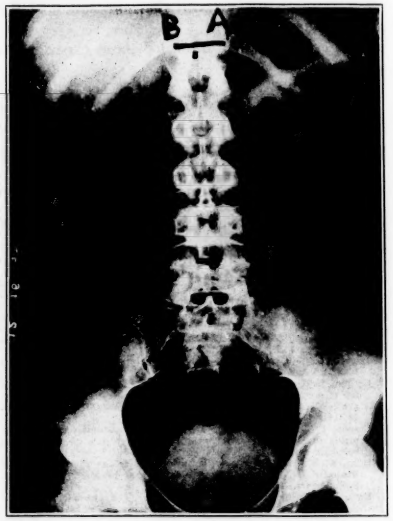

Figure 7. *The Effect of Benzedrine Sulfate (20 mg. intravenously) and Atropine (0.9 mg. intravenously): One hour after injection. Note the dilated bladder.*

The addition of Prostigmin to Mecholyl definitely enhances the action of the latter drug on the entire urinary tract. Theoretically this synergistic action is due to the choline-esterase-destroying property of Prostigmin, so that the Mecholyl is allowed to act more effectively. Atropine in large effective intravenous doses results in dilatation of the urinary tract, theoretically by diminishing parasympathetic activity so that sympathetic activity becomes predominant. It questionably enhances the effect of Benzedrine Sulfate tending to cloud the roentgenographic changes in the upper urinary tract. A questionable synergism of these two drugs has also been observed in studies on the gastrointestinal tract.

From the observations of the present study, certain clinical applications of these drugs are suggested. Mecholyl has been used by many workers to stimulate the atonic bladder.[9, 10, 11, 12] Unfortunately the drug is relatively inert when taken by mouth, even in very large doses. In 10-mg. subcutaneous doses, which cause a relatively mild systemic reaction, one of us (J. L.) has observed effective stimulation of micturition in 2 cases of atonic bladder which complicated the convalescence of grippe. Doryl, a related choline, has a like effect on the atonic bladder.[13, 14]

Unlike Mecholyl, Benzedrine Sulfate is very effective by mouth and thus may be useful in the clinical field of urinary disorders. The fact that this drug causes the kidney to stand out sharply following intravenous urography might make its

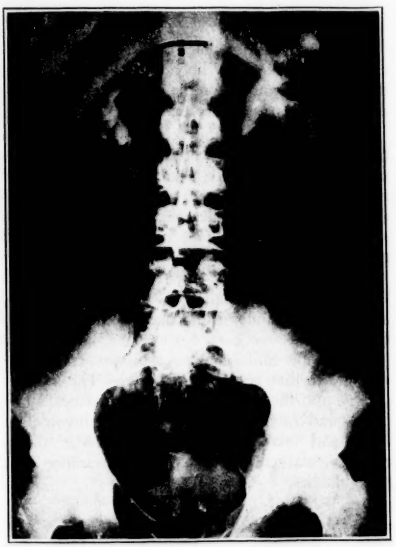

Figure 8. *The Effect of Atropine (1.2 mg. intravenously): Forty-six minutes after injection. The kidney is better visualized than in the control plate; the pelves, calyces, ureters and bladder show moderate dilatation.*

administration useful in detecting abnormalities of the adrenals which result in distorting the kidney. Another use of Benzedrine Sulfate might be in cases of ureteral stone in order to cause relaxation, so that the calculus might pass down the ureter to the bladder more quickly. The drug might also be useful in spasm of the ureter due to other causes than calculus. Another suggested use of Benzedrine Sulfate is for the small, contracted bladder due to tuberculosis or other causes. One of us (B. G.) has followed a case of

tuberculous bladder for several months which was characterized by marked pain, tenesmus and frequency, usually every half hour. This patient was given 30 mg. of Benzedrine Sulfate by intramuscular injection. Immediately before the administration of the drug the capacity of the bladder, measured by the amount of sodium iodide solution that could be tolerated, was only 60 cc. A marked change in the capacity of the organ occurred following the administration of the Benzedrine Sulfate, so that within half an hour the patient was able to tolerate 180 cc. of sodium iodide solution in the bladder. Definite diminution in the patient's urinary symptoms has continued for several months following the taking of Benzedrine Sulfate by mouth.

SUMMARY AND CONCLUSIONS

Intravenous urographic studies following the administration of Mecholyl, Prostigmin, Benzedrine Sulfate and atropine were done on 6 patients with dementia praecox.

Mecholyl causes definite changes in the urinary tract. The kidneys diminish in size; the pelves and calyces become narrowed; the ureters become contracted and show marked peristalsis; the bladder contracts markedly, becomes triangular in shape with flattened top, and shows increased trabeculation.

Prostigmin enhances the effects of Mecholyl. Used alone in the small doses we have employed, it causes no definite visible changes in the urinary tract.

Benzedrine Sulfate gives a picture completely opposite to that given by Mecholyl. The kidneys enlarge and their general visibility increases. The pelves and calyces dilate; the ureters become relaxed and increase in size; the bladder shows marked enlargement, giving the appearance of a cord bladder.

Atropine sulfate in large intravenous doses causes dilatation of the pelves, calyces and ureters, and increases the visibility of the kidneys, but definitely less so than does Benzedrine Sulfate. When it is combined with Benzedrine Sulfate, the upper urinary tract becomes obscure; the bladder becomes dilated, in some cases more markedly than with Benzedrine Sulfate alone.

For pharmacological studies the technic of intravenous urography is obviously definitely superior to the retrograde methods which appear to interfere mechanically with the pure action of the drug studied.

Some clinical applications of these experiments are suggested.

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PAPERS FROM THE FAULKNER HOSPITAL

ACTIVATION OF THE PERITONEAL MECHANISM OF DEFENSE

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IN a recent paper Harrison[1] states: "It is a mistake to think of any of our operative wounds as 'sterile,' that is, as completely uncontaminated by micro-organisms. In all of them the resistance of the tissues is pitted against a certain amount of contamination, and if the contamination exceeds this degree of resistance infection results." This observation has long been accepted as a surgical axiom, and in this paper we are chiefly concerned with its application to the peritoneal cavity. A gradual trend toward the use of peritoneal "immunization" suggests the desirability of a brief review of the history of this effort and of recent observations on its clinical application.

As early as 1894 Issaeff and Ivanoff[2] were able to protect experimental animals against infection of the peritoneum with cholera vibrios by previous intraperitoneal injection of various substances, namely water, serum, urine, nucleic acid, bouillon and tuberculin. In 1897 Durham[3] protected experimental animals against infection of the peritoneum with *Bacillus typhosus* by the previous intraperitoneal injection of sodium chloride. Solieri[4] in 1902 protected guinea pigs against *B. coli* peritonitis by previous intraperitoneal injection of sodium chloride. In 1904, von Mikulicz[5] and Miyake[6] used nucleic acid, and vaccine made from *B. coli,* to protect rabbits from fecal peritonitis. The same year, von Mikulicz[6] used nucleic acid and sodium chloride intraperitoneally in a small series of patients. At this time he also tried the clinical application of a vaccine made from *B. coli,* but with such distressing reactions that he was forced to abandon the practice.

In 1922 I[7] first used amniotic fluid intraperitoneally following surgical procedures in an effort to stimulate the processes of defense and repair in the peritoneal cavity. From 1922 to 1927 it was necessary to confine this work to abdominal cesarean sections, since the only sterile amniotic fluid available was that obtained at operation from cases uncontaminated by vaginal examination or rupture of the fetal membranes. In 1927, however, a commercial product, amniotic fluid concentrate, made from bovine amniotic fluid, became available, and through its use the scope of this technic was widened to include surgery of all serous cavities.

Steinberg and Goldblatt[8] in 1927 reported on the protection of a large series of dogs against fecal peritonitis by the previous intraperitoneal injection of a vaccine made from *B. coli.* Again in 1933 these investigators[9] reported on a similar series of experiments in which they claimed to have achieved better results by suspending killed *B. coli* in a 1 per cent solution of gum tragacanth. Rankin and Bargen[10] in 1929 announced the intraperitoneal use of a vaccine made from *B. coli* and streptococci. They used this forty-eight to seventy-two hours previous to bowel resection and claimed a very striking reduction in their operative mortality. The peritoneal cytologic response to this vaccine was reported on by Dixon and Rixford[11] in 1934, and one year later Dixon and Bargen[12] published results on its clinical use.

Young and Marks[13] in 1934 reported on the preoperative intraperitoneal injection of amniotic fluid concentrate in cases of resection of the colon. In a combined clinical and experimental study they concluded that the injection of this substance into the peritoneal cavity six to eight hours previous to operation created a sterile peritonitis which, at the time of opening the abdomen, varied in degree from a mild injection and a thin fibrinous coating of the peritoneal surfaces to a thick plastic exudate with a few ounces of a fluid exudate in the deep recesses of the pelvis. The white cell count of the blood averaged 25,000 and that of the exudate 50,000. This result was not unlike that obtained in the use of the bacterial vaccines, except that fibrin was more abundant, the time interval required for maximum protection was much shorter, and the physiologic upset attendant on the use of the bacterial vaccines was absent. In connection with the employment of this substance they noted a striking reduction in the mortality from postoperative peritonitis. In 1936 we[14] reported the results of a comparative experimental study on dogs of the peritoneal cytologic response to the use of *B. coli,* amniotic fluid concentrate, normal salt solution, Merthiolate solution (1:20,000) and sodium ricinoleate solution (1 per cent). Weighing the advantages and disadvantages of all the substances used, it was concluded that amniotic fluid concentrate gave the most protection with the least local and systemic disturbance.

From the foregoing brief review of the persistent and varied attempts to activate the peritoneal defense mechanism by the intraperitoneal injection of a sterile substance, it would seem that

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a necessity for additional protection beyond that afforded by a careful operative technic has been recognized.

An ideal activator of the mechanism of peritoneal defense should be sufficiently vigorous to be effective and yet not cause gross irritation or cell destruction. It should have an immunizing interval sufficiently brief to make it adaptable to operative as well as preoperative introduction. Its protecting effect should last long enough to exceed the danger period of the development of infection from operative contamination.

The three substances now in clinical use as activators of the peritoneal defense mechanism are: amniotic fluid concentrate (Amfetin), Rankin and Bargen's *B. coli*-streptococcus vaccine and Steinberg's *B. coli* vaccine (Coli-Bactragen). Extensive clinical and experimental trial of these agents has furnished considerable information as to their respective effects. The investigations by Rankin and Bargen,[10] Dixon and Rixford,[11] Dixon and Bargen,[12] Young and Marks,[13] Coller and Ransom,[15] Potter and Coller,[16] Morton,[17] Herrmann,[18] Seeley and his associates[19] and us[14] have given sufficient proof of the value of this new technic in clinical operative procedures. Further proof of the growing popularity of the clinical use of these substances is furnished by Ravdin and Johnston,[20] who state in a recent paper that "preoperative vaccination of the peritoneum in patients without acute obstruction is becoming a more accepted procedure."

With the establishment of peritoneal stimulation as a clinical procedure the surgeon is confronted with the necessity of choosing an activating agent. The bacterial vaccines and amniotic fluid concentrate each have their enthusiastic sponsors.

Coller and Ransom,[15] writing concerning carcinoma of the rectum, say:

> In all of the patients on whom the one stage operation has been performed the peritoneum has been protected against infection by the introduction of bactrogen [*sic*] as developed by Steinberg. . . . The effect produced is not specific but is entirely an hyperleucocytosis. . . . We feel that the use of this substance is worth while to guard against contamination that occurs not infrequently in the operations we have performed.

Steinberg[21] says of the local and systemic reactions to his vaccine (Coli-Bactragen):

> Abdominal pain and distention followed by nausea and occasional vomiting appeared approximately two and one half hours after peritoneal injection. The temperature usually rose 4 degrees with a corresponding rise in pulse rate. At the height of fever the patient had a chill. These symptoms were occasionally entirely absent or varied in degree and disappeared in 24 to 48 hours.

Steinberg's observations were confirmed by Potter and Coller.[16]

Kimpton,[22] having used amniotic fluid concentrate in over 2000 abdomens, says:

> I do not use it preoperatively because it is my impression that sufficient mobilization of the peritoneal defense mechanism takes place within a few hours to give adequate protection against the development of a fatal peritonitis in cases of operative contamination. Its injection before closing the cavity is now a matter of routine in my abdominal and joint work. An additional factor, of great value, is the smoothness of convalescence in cases where this substance has been employed. I rarely use less than 100 cc., except in small children or joint cavities, and I frequently use up to 500 or 800 cc. in very critical cases. I have never seen any immediate or remote untoward effects from the use of this agent.

Findlay,[23] referring to his use of amniotic fluid concentrate as an activator of the peritoneal defense mechanism preoperatively, says:

> A preliminary injection of a concentrate of amniotic fluid, advocated by Young, is a simple, rapid, nonhazardous and effective method of increasing peritoneal resistance. This procedure promises to supplant the more troublesome, painful and time-consuming method of introducing colon bacilli and streptococci as an intraperitoneal vaccine, with which Rankin and Bargen so ably pioneered. I have injected amniotic fluid many times without the slightest discomfort to the patient. Rarely, the fluid spreading throughout the peritoneal cavity has caused slight nausea, but many times the patient has not been fully aroused from sleep by the procedure. Within a few hours there is usually a rise in temperature of 1° or 2°F., accompanied by a leucocyte count from 15,000 to 20,000 and an increase in the leucocyte count of the peritoneal exudate, which becomes turbid.

All investigators agree that the primary local leukocytosis which follows the intraperitoneal injection of any of the agents under discussion is a major factor in the defense against the development of a fatal postoperative peritonitis. Rankin and Bargen[10] and Dixon and Rixford,[11] noting the clinical effect of their *B. coli*-streptococcus vaccine, agree substantially with observations made by Steinberg and Goldblatt,[9] Coller and Ransom[15] and Potter and Coller[16] on the clinical use of Coli-Bactragen. The only real exception to this is a difference of opinion on the character of the cytologic response evidenced in the exudate, Steinberg claiming a predominance of polymorphonuclears at the time of maximum protection, while the former group claim a preponderance of histiocytes at the peak of local immunity. Seeley and his associates[19] at the Mayo Research Foundation recently observed in an experimental study of the peritoneal cytologic response to equal quantities of amniotic fluid concentrate, Bargen's vaccine and a 2 per cent solution of sodium ricino-

leate that "as between animals receiving amniotic fluid and those receiving vaccine, there was no outstanding difference." Another observation made by Seeley and his associates in this study, the significance of which is quite obscure, is that while the histiocyte count of the exudate reached its maximum at the end of three days in the animals receiving vaccine and sodium ricinoleate, the animals receiving amniotic fluid concentrate showed a continued rise to the end of the experiment, approximately one week. In view of the fact that Dixon and Rixford,[11] Dixon and Bargen[12] and others have consistently regarded the number of histiocytes as a measure of the protection conferred by intraperitoneal vaccination, further study of this persistent rise of histiocytes in the exudate of animals given amniotic fluid may throw some light on the role of these cells in the development of resistance to infection of the peritoneal cavity. If protection were to be measured by the leukocyte response in the exudate immediately following intraperitoneal injection, we should be forced to conclude that the bacterial vaccines give the best results. However important the peritoneal cytologic response may be in the prevention of peritonitis, the value of fibrin in this defense mechanism cannot be ignored. That it is a most important defense element is attested to by Buchbinder,[25] who says: "Rapidity of absorption in the largest measure governs the prognosis in acute diffuse peritonitis, and . . . fibrin is the most important factor in controlling the rate of absorption." Since the protection afforded by intraperitoneal injection of bacterial vaccines or amniotic fluid concentrate is chiefly local, such protection in order to be most effective should simulate the "fixation" described by Menkin.[24] Without an abundance of fibrin, fixation is obviously less effective. The exudate induced by the intraperitoneal injection of amniotic fluid concentrate is much richer in fibrin than the exudate produced in response to the use of the bacterial vaccines. This observation has been confirmed by Dixon and Bargen.[12]

As previously stated, it is generally agreed by all investigators that the primary local protection in the use of these activating agents is nonspecific. This is contrary to the early conclusions of those working with *B. coli*-streptococcus vaccine. It was their impression that the chief advantage of the use of a bacterial vaccine was specific immunity. This, of course, accounts for their selection of the streptococcus and *B. coli* in making a vaccine, since these bacteria have long been regarded as common agents in most cases of postoperative peritonitis. With the waiving of any claim to specific local peritoneal protection by the one-injection method, preoperatively or postoperatively, there remains practically no advantage in the use of bacterial vaccines over a less disturbing substance if the latter can produce substantially the same cellular response. Observations of Seeley and his associates[19] have shown that this is true for *B. coli*-streptococcus vaccine and amniotic-fluid concentrate. Repeated intraperitoneal injections of the bacterial vaccines over a number of days may establish a systemic immunity in which the peritoneum shares. The practice of repeated intraperitoneal injections, however, is not popular because of the local and general upset and the time consumed. Neither can it furnish specific immunity against any but the bacteria from which the vaccine is made. In the average case of postoperative peritonitis many bacteria other than the streptococcus and *B. coli* are involved.

There is likelihood that continued research will bring to light other agents that are as potent as the bacterial vaccines and amniotic-fluid concentrate in producing an effective peritoneal defense. Up to the present, at least, no other substances have been subjected to such extensive experimental and clinical trial. Until some positive yardstick of peritoneal protection is established, the foregoing experimental and clinical observations on the use of these two agents will have to serve as a measure of their comparative value and of the value of peritoneal protection in general.

SUMMARY

Attempts to protect the peritoneum against postoperative infection cover a period of over forty years and have established the fact that the introduction of certain non-infectious substances into the peritoneal cavity produces a reaction which is of definite assistance in preventing the effects of operative contamination. Amniotic-fluid concentrate and bacterial vaccines are the activating agents most generally employed.

A comparative evaluation has been attempted of the bacterial vaccines and amniotic-fluid concentrate on the basis of peritoneal cytologic response, fibrin content of the exudate, length of immunization interval, protection accomplished and the physiologic upset attendant on their use. Extensive clinical and experimental trial suggests strongly that the use of these substances is of definite value in combating the morbidity and mortality from postoperative peritonitis.

No positive measure of peritoneal protection other than experimental and clinical observation is at present available.

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CASE RECORDS OF THE FAULKNER HOSPITAL

Antemortem and Postmortem Records as Used in Monthly Clinicopathological Conferences

Directed by J. Beach Hazard, M.D.

CASE 6385

Presentation of Case

First Admission. A fifty-year-old, American saleswoman was admitted with the complaint of pain in the upper abdomen.

About one month preceding admission the patient experienced pain and discomfort, starting in the hypogastrium and later localizing in the epigastrium. This lasted for a week and was not accompanied by nausea or vomiting. Four days before entry she was suddenly seized with epigastric pain and discomfort, accompanied by distention and belching. These symptoms increased in severity and were accompanied by chills and fever. There was no nausea or vomiting, no radiation of pain to the shoulder or scapula and no jaundice or abnormal stools. The patient was menstruating at the time of both the above-mentioned attacks.

Her general health had always been good. She had had the usual childhood diseases, including scarlet fever and pertussis. She had had a miscarriage twelve years before entry in the second month of pregnancy. Weight had been constant at around 160 to 165 pounds. Catamenia began at eleven years, were regular at twenty-eight-day intervals and lasted seven days; flow was of normal amount.

The patient's father died at the age of fifty of "fatty degeneration of the heart." Her mother died at sixty-five of carcinoma of the breast. A paternal grandfather died of carcinoma of the stomach. Her husband was living and well, and there were no children.

Physical examination revealed a well-developed and well-nourished woman lying in bed, obviously distressed. The abdomen was obese and somewhat distended, and there was marked tenderness in the upper portion, especially in the epigastrium. No masses, spasm or signs of fluid could be elicited. The heart and lungs were negative. Pelvic and rectal examinations were not done. The temperature on admission was 100.2°F., pulse 100, respirations 24 and blood pressure 130 systolic, 90 diastolic.

A catheterized specimen of urine showed an acid reaction, a specific gravity of 1.014, a slightest possible trace of albumin, no sugar and no bile, and the sediment contained from 10 to 20 red blood cells, 5 to 10 leukocytes, and 1 to 5 hyaline casts per high-power field. The blood showed a white-cell count of 7000 with 75 per cent polymorphonuclears, and a red-cell count of 4,200,000 with a hemoglobin of 84 per cent (Sahli). Stool examination was negative. A blood Hinton test was negative.

Soon after admission ½ gr. of codeine was given, after which the pain subsided and did not recur during the four days of the patient's stay in the hospital. On the second day the temperature re-

turned to normal and remained so until discharge.

Second Admission (one month later). The patient remained quite well for three weeks after leaving the hospital, and then began to feel generally out of sorts and developed a sensation of stiffness in the lower abdomen. These symptoms at first were not sufficiently severe to induce her to restrict her activities, but during the fifth day before entry they increased to the point of incapacitating her for her housework. At this time she developed pain with urination, which radiated to both lower quadrants and was associated with tenesmus and frequency. Three days before admission her menstrual period started, and coincidentally she noted the onset of severe, steady pain, mostly in the right lower quadrant but also somewhat in the right upper quadrant and epigastrium. The pain increased in severity and became somewhat localized to the right lower quadrant.

Physical examination revealed a woman with a flushed, moist countenance. The abdomen was poorly relaxed throughout and was generally tender, but in the right lower quadrant and extending up into the right upper quadrant there was exquisite tenderness so that even the slightest pressure caused pain. There was marked voluntary spasm, and it was impossible to say if there was any involuntary component. No evidence of masses or fluid could be elicited. There was a suggestive icteric tint to the sclerae. Pelvic examination was attempted but was not successfully done because of extreme discomfort. The examination otherwise was as stated on her previous admission. The temperature was 99.2°F., the pulse 102, respirations 22 and the blood pressure 130 systolic, 85 diastolic.

A single catheter specimen of urine showed an acid reaction, a slightest possible trace of albumin and no sugar; the sediment contained from 18 to 20 red blood cells and 5 to 10 leukocytes per high-power field. The white-cell count of the blood was 12,700 with 83 per cent polymorphonuclears. The red-cell count was 4,600,000 with a hemoglobin of 92 per cent (Sahli). The icteric index was 10. A stool examination was negative.

After admission the pain was controlled by morphine and codeine; the temperature for three days following entry varied from 97 to 100.5°F. At the end of the third day an abdominal operation was performed.

During the postoperative course the temperature ranged up to 99°F., but on the fifteenth day after admission became normal and so remained. Until near the time of discharge the patient continued to have cramp-like pain following meals; tincture of belladonna was given. She improved steadily and gained strength, so that when leaving the hospital she had had a normal temperature for five days. During the two months since discharge she has been free of all symptoms.

Differential Diagnosis

Dr. Charles P. Sheldon: In summary, we have a woman fifty years old whose symptoms are largely localized in the epigastrium and in the right lower abdominal quadrant and who has had three attacks of abdominal pain coming at the time of menstruation, has had mild fever and is sterile. Among the diagnoses to consider are gall-bladder disease, appendicitis, diverticulitis, ureteral obstruction, ovarian cyst, pelvic inflammatory disease with perihepatitis, endometriosis, pleurisy, coronary disease, gastric crises and carcinoma.

Against gall-bladder disease is the absence of shoulder pain and jaundice. The history and the time interval contradict a diagnosis of appendicitis which either subsides or goes on to perforation and peritonitis or abscess formation within five to eight days. The patient does not seem to have been sick enough to have had an appendiceal abscess. The bowel habits were normal, as were the stool examinations, so that diverticulitis is unlikely.

At both admissions there were erythrocytes in the urine. This brings up the possibility of calculus in the urinary tract, ureteral stricture or some diseased process in the bladder. Since there is a possibility of endometriosis there may be an endometrial implant in the bladder wall. The distribution of pain is not typical for ureteral calculus or stricture.

A diagnosis of ovarian cyst cannot be made as no tumor was felt abdominally or was palpable by bimanual examination. The negative Hinton test is against gastric crises due to lues. There is a history of carcinoma in the family, but the patient's general health had always been good and there had been no loss of weight. Examination of the heart is reported as normal, and there is no positive support for the diagnosis of coronary occlusion.

The fact that the patient was menstruating at the time of the attacks of abdominal pain suggests two abdominal conditions that are likely to flare up at the time of the menstrual period: pelvic inflammatory disease and endometriosis. It is impossible to make a diagnosis of endometriosis except by pelvic examination and it is certainly not possible to diagnose an endometrial implant in the bladder wall without performing a cystoscopy. The presence of fever at both hospital admissions is contrary to a diagnosis of en-

dometriosis. The bladder irritability and the story of abdominal pain made worse by the menstrual period suggest pelvic inflammatory disease. Tenderness in the right upper quadrant and in the epigastrium could be explained by an associated perihepatitis.

I shall digress a moment on the subject of perihepatitis. A few years ago Curtis[1] described the violin-string adhesions found on the upper surface of the liver associated with pelvic inflammatory disease, particularly of the gonorrheal type. A differential diagnosis of pain in the right upper quadrant of the abdomen should always include consideration of adhesions to the anterior surface of the liver. If the cause of this pain is obscure, pelvic examination may be valuable in differentiation. On the other hand, in the presence of a pelvic inflammatory mass of unknown etiology, the history of pain in the right upper quadrant may be of value in determining the etiology of the pelvic lesion.

This patient had three attacks of abdominal pain associated with menstruation. In the first attack, pain started in the hypogastrium and later settled in the epigastrium; in the second it started in the epigastrium; in the third it started in the right lower quadrant of the abdomen and radiated upward into the right upper quadrant. The fact that she had so much pelvic tenderness that it was impossible to perform a satisfactory pelvic examination is in agreement with a pelvic inflammatory lesion.

There are some lesions which simulate gonorrheal disease. One thinks of postabortive conditions, but there is no history of an abortion and she had had menstrual periods regularly. There is also the question of tuberculosis of the fallopian tubes, which comprises about 5 per cent of the cases of salpingitis, but it is impossible to distinguish between tuberculosis and severe gonorrheal salpingitis. Graves[2] has pointed out the association between appendicitis and salpingitis. Tubal infection due to organisms such as *Bacillus coli* and the staphylococcus usually results in pyosalpinx, but this cannot be corroborated in this case because there was not a satisfactory pelvic examination. Ectopic pregnancy is to be considered, but would be ruled out by the patient's age and the regular occurrence of menstrual cycles.

In chronic pelvic inflammatory disease the symptoms and signs depend on the structural lesions. Dysmenorrhea is frequent. There is usually pain before and throughout the flow, which is true in this case. Abnormal periods are frequent. From 35 to 50 per cent of the cases of pelvic inflammatory disease have frequent and profuse periods. Leukorrhea is usually present. The patient experiences pain on one or both sides of the lower abdomen, which is increased by exercise and constipation. Before the last admission, exertion definitely aggravated this patient's symptoms. Vesical irritability is common, and was present in this case.

To be positive of the diagnosis, I should like to know the results of a satisfactory pelvic examination. Since this, however, is not possible, I shall base my conclusions on the data given and say that the patient has pelvic inflammatory disease with perihepatitis.

Dr. Edward L. Young, Jr.: On the first admission the most likely diagnosis seemed to be gall-bladder disease. The patient had very definite spasm over the gall bladder when first seen, and later definite tenderness. At the second admission she had had symptoms for three days. She was a definitely sick woman, and when I felt her abdomen I was alarmed and thought she had appendicitis, with possibly a generalized peritonitis. Immediately after she came in, the first pelvic examination was made; it was unsatisfactory, with merely the suggestion of what I interpreted as an appendiceal abscess. She then quieted down very promptly. A second pelvic examination revealed a definite, even mass in the right side of the pelvis.

The operation showed the lesion to be in the right lower quadrant, and to consist of an infected cyst, with a localized peritonitis. The patient returned to work and has been perfectly normal for the four months after leaving the hospital; inasmuch as there were no symptoms of gall-bladder disease and no recurrence of pain, a Graham test has not been done.

Clinical Diagnosis

Pelvic abscess, secondary to appendicitis or salpingitis.

Dr. Sheldon's Diagnosis

Chronic pelvic inflammation, with perihepatitis.

Anatomical Diagnoses

Simple dermoid cyst, with chronic and recurrent acute inflammation.

Pathological Discussion

Dr. J. Beach Hazard: The specimen removed at operation consisted of a dermoid cyst 10 cm. in diameter and filled with hair and dark-yellow material. The surface varied from red to gray and presented occasional patches of fibrin. The wall was 0.5 cm. or less in thickness.

Dr. Henry C. Marble: If the patient had had an x-ray of the abdomen do you think a diagnosis could have been made?

DR. HAZARD: Neither bone nor teeth were present, so that it is doubtful if an x-ray film would have aided in the diagnosis.

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CASE 6387

PRESENTATION OF CASE

A sixty-six-year-old, German-American housewife was admitted with the chief complaint of jaundice.

Three and a half months preceding entry the patient had had a mild attack of shingles, followed by jaundice that had gradually increased. Two months before admission she was admitted to a hospital where, she stated, x-ray films were taken which showed no evidence of disease in her stomach. At this time a mass was noted between the gall bladder and the liver, and she was told that she must have an operation, but would not consent to it. One and a half months before entry she went to her family physician, who put her on a fat-free diet. Her jaundice cleared up slightly following this, but soon returned. One month preceding admission she began to have severe itching. She was given a white wash by a skin specialist, which did not relieve her symptoms. The itching became more severe. About four days before entry she suddenly became weak and drowsy and had difficulty in standing up. The stools had been clay colored since the jaundice began and the urine had been very dark. The bowel movements had been regular with the aid of mineral oil. There had been slight bleeding from the rectum, apparently from hemorrhoids. There was no pain at any time. The patient had lost about 15 pounds since the onset of her illness.

The past history revealed no operations. She had had scarlet fever. She had had rheumatism in the joints of her extremities for years. Her appetite had always been good. Her husband died of an unknown cause. One son and one daughter were living and well.

Physical examination revealed a well-developed, obese woman lying in bed, apparently in no pain. She was very drowsy and responded only to direct questions. The skin was markedly jaundiced over its entirety and was covered with minute red abrasions. There was no tenderness over the sinuses or mastoids. The pupils were equal and regular and reacted to light and accommodation. The conjunctivae were definitely yellow and presented no petechiae. The ears and nose were negative. All teeth were absent with the exception of four in the upper jaw. The mouth, tongue and throat were otherwise negative. There was no glandular enlargement. The thyroid was not remarkable. The lungs were clear throughout. The heart was not enlarged and the sounds were normal. No murmurs were present. The rate and rhythm were normal. The liver was palpable about two fingerbreadths below the right costal margin. There was a suggestion of an irregular mass just below the liver and to the right of the midline in the epigastrium. There was no tenderness or spasm. Rectal examination revealed some external hemorrhoids. The extremities were negative. The temperature was 98.4°F., pulse 84, respirations 26, and blood pressure 128 systolic, 76 diastolic.

Urinalysis showed a hazy amber specimen of acid reaction, with a specific gravity 1.020, a slight trace of albumin, no sugar, a positive test for bile, and a sediment that showed no red blood cells or casts but 3 to 4 white blood cells per high-power field and many epithelial cells. The icteric index was 100. One stool was clay colored and showed no occult blood. The red-cell count of the blood was 3,700,000, and the white-cell count 5950. The hemoglobin was 83 per cent (Sahli). A differential count showed 71 per cent polymorphonuclears, 1 per cent large lymphocytes, 21 per cent small lymphocytes, 5 per cent monocytes and 2 per cent eosinophils. The red blood cells and platelets appeared normal. The blood-clotting time was 50 minutes. A blood Hinton test was negative.

After three days' stay in the hospital the patient's drowsiness disappeared. It was decided that it had probably been due to sedatives given before entry. She seemed mentally alert. She felt well at this time except for severe itching of the skin. Ergotamine tartrate was used and gave some relief. The temperature remained between 97 and 100°F. The Takata-Ara test was negative on two occasions. A van den Bergh reaction (direct) showed 10.7 mg. per cent of bilirubin.

On the seventh hospital day the abdomen was opened through a gall-bladder incision. The liver was hard and purplish red. The gall-bladder and bile ducts were of average size. No tumor mass could be felt. The common duct was opened and was found to contain a slight trace of bile. A probe could be introduced into the duodenum, and no obstructions or stones were felt. A probe was passed upward into the ducts in the liver for about 12 cm., with no apparent obstruction. A biopsy was taken from the liver edge.

Following operation the patient's blood pressure

dropped to 54 systolic, 40 diastolic. Intravenous fluid was given, and she progressed satisfactorily for two days. On the night of the second postoperative day she began to bleed from the wound, but this was apparently controlled by thromboplastin applied locally and 5 cc. of Congo red administered intravenously. The coagulation time, however, was found to be prolonged to 2 hours and 10 minutes; placental extract was administered in two doses at fifteen-minute intervals, and at the end of one hour the coagulation time was halved. Liberal doses of morphine were given for restlessness. Suddenly, early on the third postoperative day, the respirations became labored and shorter and the pulse weaker. The patient went downhill rapidly and died.

Differential Diagnosis

Dr. Arthur R. Kimpton: As we study the history of this patient it is apparent that she had a gradually increasing jaundice and a mass in her abdomen which was probably the liver edge. It suggests a slowly progressing jaundice due to biliary obstruction. The fact that she had no pain coincides with the gradually increasing enlargement of her liver; the gradual increase in size of an organ over a long period of time does not give pain, whereas a sudden enlargement causes pain. Normal pupillary reflexes and the negative blood Hinton test would be points against syphilitic cirrhosis. The possibility of parasitic infection is to be considered, but no basis for such a diagnosis can be found in the history. The blood pressure is a little low for the age, which could be explained by the retention of bile salts, or perhaps some myocardial changes. Her drowsiness and weakness make me wish to know about the specific history of drugs. I speak of that with emphasis because I recently brought a patient into the hospital with a history simulating intestinal obstruction, uremia or diabetic coma, and it was later discovered that the patient had been taking Amytal. As this patient's drowsiness disappeared after three days' stay in the hospital, her mental lassitude was not due to the jaundice or it would undoubtedly have remained.

The high icteric index is indicative of biliary retention, and the clay-colored stool is, of course, more positive evidence, though it does not localize the point of obstruction. Acute yellow atrophy can be discarded because of the size of the liver. Biliary obstruction seems more likely. As to the Takata-Ara test, I have not much faith in it. The direct van den Bergh reaction and high-serum bilirubin are helpful in arriving at a diagnosis. I think because of this direct test one must take into consideration hepatogenous jaundice with obstruction.

The appearance of the liver noted at operation merely indicates biliary retention. Certainly it is not a liver of hemachromatosis, which is usually brown, and it is not one of acute yellow atrophy, which is usually contracted and pinkish, even though it may show temporary enlargement. That the gall bladder and bile ducts were of average size is important in localizing the biliary obstruction, because this certainly indicates that it was not at the papilla. Up to this point we can say that if there was biliary obstruction it must have been above the common duct.

In regard to the therapy for the bleeding, Congo red in small doses, as I understand it, is supposed to lower the coagulation and bleeding times, but if too much is given it will do the opposite. Thromboplastin has not been of much value to me, and certainly with this type of bleeding I doubt if there is anything that will stop it. With liver disease such as this I believe that the hemorrhage is not due to interference with calcium metabolism but is associated with the retention of bile salts.

To me the whole history is definitely that of biliary obstruction. The patient had lost 15 pounds. She was in the cancer age. She had no history of parasites which might obstruct her ducts. She had had a gradual onset of her jaundice without pain, which is suggestive of gradual new growth somewhere in the biliary tree. The van den Bergh reaction is very suggestive of mechanical obstruction. Furthermore, at operation an undilated common duct and an undilated gall bladder without stones were found. The liver was not that of hemachromatosis, acute yellow atrophy or syphilis. Nor was it that of chronic passive congestion, because there would in that event have been a history of edema, probably with pain, and there would not have been the deep jaundice that this patient had. I may add that the biopsy showed nothing except bile retention.

The diagnosis as I see it is carcinoma in the hepatic ducts of the biliary tree either one or both and probably near their junction. It is a rare disease, but I cannot make any other diagnosis.

Clinical Diagnoses

Hepatitis.
Carcinoma of biliary tract?

Dr. Kimpton's Diagnosis

Primary bile-duct carcinoma.

Anatomic Diagnoses

Primary bile-duct carcinoma with obstruction of the hepatic bile ducts.
Obstructive cirrhosis.
Icterus.
Bile nephrosis.

Pathological Discussion

Dr. J. Beach Hazard: The piece of liver that was removed for biopsy showed obstructive cirrhosis. At autopsy the liver was enlarged and dark green. The common bile duct was of average caliber. The hepatic ducts at the point of junction were obstructed by a tumor mass arising just posterior to and extensively invading the right radicle and to a lesser extent the left. The tumor tissue was necrotic and very soft so that a probe passed readily through it. Histologically the architecture was that of a bile-duct carcinoma. The wound contained about 50 cc. of blood clot, and the peritoneal cavity approximately 100 cc. of bloody fluid. There was marked icterus and the kidneys showed a bile nephrosis.

A Physician: I am interested in the statement that the patient had shingles shortly before the onset of the jaundice. We know that certain cases of malignant disease which involve the vertebral column may cause herpes zoster. Was there any evidence of metastases in the spinal column?

Dr. Hazard: Only the bodies of the lower thoracic and the lumbar vertebra were examined, but these showed no tumor.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24431

PRESENTATION OF CASE

A seventy-one-year-old native, single male entered complaining of weakness, anorexia, diarrhea and insomnia of three weeks' duration.

For twenty years the patient had known that he had "leaking valves" in his heart. During that time he had occasional twinges of sharp pain over the precordium, sometimes radiating down the left arm. These lasted only a few minutes. Less frequently he noted palpitation and an irregular rhythm. For the past five or ten years he had been forced to go to bed two to three days at a time every few months because of difficulty in breathing. He nevertheless considered himself in good health and enjoyed moderate activity until six months before admission. At that time he noted "sticking" pains in his legs and an aching pain in his right shoulder. He was not certain but thought that he remembered soreness and redness of the knee joints at that time. Six weeks before entry he had a brief spell of difficult breathing and went to bed for two days. Beginning shortly thereafter he lost strength rapidly. During the year before entry he had had loss of vision with blurring and for six months had had dizzy spells, but these had grown worse during the past month. Four weeks prior to admission he lost his appetite, became slightly nauseated and felt very weak. Loss of hearing and tinnitus developed. He also noted steady aching pains in the shoulder blades and intermittent brief mid-abdominal pains made worse by coughing. Three weeks before entry he began to cough. He vomited everything except beef broth and began to have six to eight liquid, dark brown, scanty bowel movements daily. Two weeks prior to admission his feet, legs and abdomen began to swell. Red spots appeared on both of his legs and soon involved most of his body. He could not sleep and had intermittent headaches. He was continuously feeling something in his throat which he tried to cough up, but unless he swallowed it, it caused him to gag and vomit. When the material was coughed up it often contained a small amount of blood. Ten days before entry he had a brisk nosebleed.

During the year before entry he had lost 25 pounds. He had had nocturia three to four times and urgency for several years. His stools had contained no blood. He was not aware of any kidney disease or abnormality of blood pressure. His family history was noncontributory.

Physical examination showed a pale, gray-haired man with puffy jowls and pitting edema of both legs, penis, sacrum and back extending almost to the shoulders. The abdomen was protuberant, the umbilicus everted. The skin was covered with purpuric areas 1 to 5 mm. in diameter, thickest over both legs and forearms. Small punctate hemorrhages were present in the folds of the skin of the palms, in the conjunctivae, on the gums, beneath the tongue and in both retinae. Pediculi were present in the pubic hair. There were early bilateral cataracts, and the fundi showed arteriosclerotic changes. The teeth showed advanced caries and pyorrhea. The breath had a very foul odor. Examination of the throat showed a hyperactive gag reflex. Bits of fresh bloody mucus were clinging to the soft palate. There were coarse moist rales over both lower lung fields extending up to the angles of the scapulae. The breath sounds were harsh anteriorly and high pitched just beneath the left coracoid process. There was questionable tenderness to percussion over that area. The heart dullness was percussed 2 cm. to the left of the midclavicular line, with the apex beat felt in the sixth interspace. The rate was 110 and regular. A harsh low-pitched systolic murmur was heard in the aortic area. A high-pitched musical systolic murmur was present in the mitral area. The second sound was practically absent. A systolic thrill was felt over the aortic area, but not over the carotid arteries. The blood pressure was 85 systolic, 55 diastolic. In the abdomen there was tenderness to deep pressure beneath both costal margins. The spleen was not palpated. The liver edge seemed to descend 1 cm. on deep inspiration. Tenderness was also elicited in the left lower quadrant. One stool was guaiac negative, two others positive.

The rectal temperature was 99°F., the pulse 110. The respirations were 20.

Examination of the urine showed a specific gravity of 1.010, a large trace of albumin, and 10 to 20 red cells, 15 to 20 white cells and an occasional granular cast per high-power field. The blood showed a red-cell count of 2,000,000 with 45 per cent hemoglobin and a white-cell count of 4000 with 76 per cent polymorphonuclears. The red cells varied slightly in size and shape and definitely in depth of staining. The platelets were reduced. The nonprotein nitrogen of the serum was 122 mg. per cent, and the serum

protein 6.2 gm. A blood Hinton test was positive, a Wassermann test anticomplementary.

X-ray films showed the heart to be enlarged on both sides. The aorta was dilated, elongated and calcified in the region of the knob. The pulmonary vessels were increased in width. There was mottled dullness in both lung fields, particularly on the right. There was evidence of fluid in both pleural cavities.

An electrocardiogram showed a ventricular rate of 100, with normal rhythm. The P-R interval was 0.19 sec. There was moderate left-axis deviation. T_1 was inverted, and P_2 notched and wide.

During the first two days in the hospital the patient was given 27 gr. of digitalis. On the third hospital day the nonprotein nitrogen of the blood serum was 165 mg. per cent. His temperature remained normal, and his pulse decreased to 80. Respirations fluctuated between 20 and 25. He rapidly failed and died on the third hospital day.

Differential Diagnosis

Dr. Howard B. Sprague: This man was seventy-one years old and therefore we should be on the lookout for cancer and for cardiovascular disease. For twenty years he was known to have had "leaking valves" in his heart. That would seem to rule out, so far as the valves are concerned, a luetic involvement which is suggested later on by the finding of a positive Hinton. It seems more likely that he had some slowly progressing degenerative change, whether or not related to previous inflammatory conditions of the valves. There is some question about palpitation and irregular rhythm. He may have had many premature beats or he may have had attacks of paroxysmal auricular fibrillation. Then, again, he had varying symptoms of congestive difficulty, which forced him to go to bed with dyspnea.

Six months before this admission he began to note pains in his legs and an aching pain in the right shoulder. These pains do not seem to have any diagnostic value. He thought that he remembered some soreness and redness of the knee joint at that time. Since it was so indefinite in his own mind, it does not make it very definite in mine.

Loss of vision with blurring and dizzy spells are symptoms that we should expect with cerebral arteriosclerosis and sclerotic changes in the eyes themselves. The same is true about the loss of hearing and tinnitus.

He began to lose his appetite four weeks before admission and became nauseated and weak. Was this merely an expression of his cardiac and vascular condition, or did he have something in the gastrointestinal tract which precipitated this final episode? He had coughing and vomiting and frequent scanty bowel movements, but there is nothing definite for a diagnosis of any process in the gastrointestinal tract. We find that later on he had one negative guaiac test on the stool and two positive ones. He had a good deal of bleeding apparently in his mouth from mucous membranes, and this could account for the latter. He began to develop signs of localized hemorrhage throughout the skin and in the mucous membranes, and he also had a nosebleed. There was a slow loss of 25 pounds in weight, apparently over a number of years. Of course arteriosclerotic individuals do lose weight, but one wonders just what the dietary situation was, particularly in relation to this hemorrhagic tendency. He stated definitely that the stools had contained no fresh blood. We must think of carcinoma of the rectum, but he did not have symptoms suggesting that unless we can say that the loose stools suggest cancer in the large bowel.

On physical examination we have evidence of hemorrhage. We have arteriosclerotic changes in the fundi and the cataracts to account for loss of vision. The cardiac findings are those of an enlarged heart, with a harsh and low-pitched systolic murmur at the aortic area, a practically absent second sound, low blood pressure and small pulse pressure. There was a thrill which could be felt at the aortic area but apparently was not propagated into the carotids, which may have been due to his condition during the last stages of his disease. All this is consistent with the symptoms of valvular disease in a man of his age, and seems to mean aortic stenosis — a diagnosis which fits in with the knowledge that for twenty years he had had something wrong with the valves in his heart. There is in addition the evidence of pitting edema, general edema and fluid in his pleural spaces but his liver was not very much enlarged.

The urinary findings do not tell us very much except for the fact that they were consistent with those of congestive heart failure, with perhaps more red cells in the sediment than one would expect.

The blood is interesting in that it showed a high degree of anemia, a low white count and some decrease in the platelets. His nonprotein nitrogen was definitely elevated and continued so to the end; this indicates that he was uremic.

Now what help do we have from the x-ray? So far as the cardiac condition is concerned, it tells us that the heart was enlarged, and the aorta elongated, dilated and calcified in the region of the knob. Such changes are consistent with an arteriosclerotic aorta, and the calcification fa-

vors this diagnosis. In the anteroposterior view the bulging may be due to the tortuosity.

The electrocardiogram shows left-axis deviation with inversion of T waves in the first lead, which may be the effect of long-continued left ventricular strain rather than any acute coronary closure. He was digitalized without much effect except that his pulse rate came down, but the nonprotein nitrogen rose and he died on the third day after admission.

This situation seems to have been due to a combination of cardiac disease and a marked hemorrhagic tendency. The story suggests scurvy so far as the blood is concerned,—the purpuric spots, the bleeding from the mucous membranes, and the anemia of this type,—and the vomiting no doubt accentuated the dietary restriction. I have seen several cases of adult scurvy; the patients have not infrequently been older men who were badly off financially and who had fallen into very poor dietary habits. They usually lived in small rooms, prepared their own food which consisted almost entirely of oatmeal or something of that sort, and eventually developed scurvy. Then there is the group of neurotic individuals who get strange ideas about diet and live on food that is inadequate in various ways. It is interesting that this man's serum protein was within normal limits. I do not know how much protein there is in beef broth, which he seems to have been able to retain, but it cannot be very much.

I am still somewhat at a loss to know what the precipitating factors were in this man's final episode. I am unable to prove that he had a malignant tumor either in the gastrointestinal tract or elsewhere. Syphilis enters the picture at least serologically. I am going to make the diagnoses of aortic stenosis, presumably calcified, and scurvy. There may be some evidence of syphilis, such as the combination of atherosclerotic and luetic changes in the aorta.

A PHYSICIAN: How about chronic glomerulonephritis?

DR. SPRAGUE: We said he was uremic so there would be a nephritic element.

CLINICAL DIAGNOSES

Arteriosclerotic heart disease.
Calcific aortic stenosis.
Congestive failure.
Uremia.
Chronic nephritis.
Symptomatic purpura.
Anemia.

DR. SPRAGUE'S DIAGNOSES

Calcific aortic stenosis.
Scurvy.
Chronic nephritis and uremia.

ANATOMICAL DIAGNOSES

Aortic stenosis, calcified.
Superimposed acute non-bacterial endocarditis.
Cardiac hypertrophy.
Purpura (? scorbutic).
Bronchopneumonia.
Chronic vascular nephritis, minimal.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: This man did have an aortic stenosis of the calcific type as Dr. Sprague suggested. His heart weighed 550 gm., not so large as we might expect with the severe degree of stenosis that the aortic valve showed. We usually argue and seldom reach a decision whether the background in these cases is arteriosclerotic, healed bacterial or rheumatic. The calcified deposits were for the most part between the cusps at their commissures rather than in the sinuses of Valsalva—a finding which suggests that the process was probably infectious rather than arteriosclerotic. To substantiate this idea there was a fair amount of calcification of the mitral valve, without any real stenosis. Some small fresh fibrinous vegetations overlay the calcified masses. They did not contain bacteria, however, either by culture or by stain. The aorta showed only a moderate degree of arteriosclerosis, and the coronaries were patent throughout. There was an extreme degree of anasarca, and the kidneys showed a moderate degree of vascular nephritis, which was not severe enough to account for the purpura on a uremic basis. The liver grossly suggested cirrhosis but histologically showed only a slight degree of scarring, too slight to qualify for a diagnosis of cardiac cirrhosis. We found evidence of bronchopneumonia throughout the lower lobes and some old tuberculosis. So far as scurvy is concerned we found nothing to prove the diagnosis but the skeleton was not examined extensively enough to rule it out. The bone marrow was quite abnormal showing diminished cellularity and hypoplasia, particularly of the white cell series. The clinicians in charge of the patient made no mention of scurvy in their diagnoses.

DR. SPRAGUE: Is there anything in the history to indicate a dietary abnormality?

DR. CASTLEMAN: It is noted that he had been keeping house for his sister and himself for the past three years, doing his own cooking. He had

meat two to four times a week, drank a quart of milk a day, had orange juice two to four times a week and for the past few months had been buying the latter by the bottle.

A PHYSICIAN: Orange juice by the bottle does not rule out scurvy. It does not contain much vitamin C.

DR. SPRAGUE: I think we can say he had subclinical scurvy in the last days of his life and that his dietary regime was disturbed. To me, it is a rather characteristic picture of adult scurvy.

A PHYSICIAN: Do you feel that that might have precipitated the final cardiac failure?

DR. SPRAGUE: I do not know. Were there any signs of syphilis?

DR. CASTLEMAN: No.

A PHYSICIAN: What other causes of purpura are there besides scurvy? Could uremia have had anything to do with it?

DR. SPRAGUE: Various toxic conditions, including uremia, resulting in capillary fragility can produce purpura.

A PHYSICIAN: How high a nonprotein nitrogen can you get with congestive failure per se?

DR. CASTLEMAN: It may go as high as 100 mg. per cent.

DR. SPRAGUE: Shall we call the kidney picture the result of simple congestive failure?

DR. CASTLEMAN: Yes. The anemia, moreover, may have been a factor in depressing the renal function.

A PHYSICIAN: Would the "sticking" pain and so forth support the clinical diagnosis of scurvy, also the pain in the joints and swelling? He never complained of joint symptoms until evidence of this scurvy began to hit him.

DR. SPRAGUE: Yes, I believe this is a point in favor of such a diagnosis.

A PHYSICIAN: There is no record as to whether the blood or urine was examined for vitamin C.

DR. CASTLEMAN: They probably would have looked for it had he lived longer. He died a few days after admission, and they did not have much chance to study the case.

CASE 24432

PRESENTATION OF CASE

A fifty-eight-year-old, native housewife was admitted complaining of jaundice of three weeks' duration.

She was perfectly well until five weeks before admission at which time she noted a gradual onset of nausea, anorexia and fatigue. These symptoms gradually increased in severity, and she consulted a physician. A gastrointestinal series was done and reported negative, and a diagnosis of "nervous indigestion" made. Three weeks prior to entry, jaundice became evident, the urine dark and the stools clay colored. She remained afebrile. The nausea continued and vomiting occurred on three occasions with emesis of green fluid. Pruritus developed over the entire body, although she had no pain at any time. She lost approximately 15 pounds following the onset of her illness. No purpuric manifestations appeared. Varicose veins were present on the right, and she developed what she called a "phlebitis" three weeks before entry.

A sister died of carcinoma of the bladder at the age of fifty-two years.

Physical examination revealed a chronically ill woman with evidence of moderate loss of weight. There was marked icterus of the skin and sclerae, and a number of scratch marks were present. Examination of the mouth showed carious teeth and pyorrhea. The lungs were negative. A soft systolic murmur was heard at the apex. The blood pressure was 140 systolic, 90 diastolic. The abdomen was doughy and moderately distended with fluid. The liver edge and gall bladder could be palpated 3 cm. below the costal margin. The liver seemed firm and slightly tender. Thrombosis of the saphenous vein was apparent on the right. There was slight, bilateral pretibial edema.

The temperature was 97°F., the pulse 100. The respirations were 25.

The urine examination showed a trace of albumin, 2 to 4 white cells per high-power field and a 4+ bile test. The blood showed a red-cell count of 4,320,000 with a hemoglobin of 80 per cent, and 13,700 white cells with 83 per cent polymorphonuclears. The serum protein was 5.5 gm. per cent, the calcium 8.45 mg., and the chlorides were equivalent to 98 cc. of N/10 sodium chloride. The bleeding time was four minutes, clotting time five minutes. The blood bilirubin content was 14 mg. per cent. A blood Hinton test was unsatisfactory. A stool examination was guaiac positive.

On the third hospital day an exploratory laparotomy was done.

DIFFERENTIAL DIAGNOSIS

DR. JOHN D. STEWART: The laboratory data in this case help me very little in making a diagnosis. The urine shows the albuminuria that one often sees in protracted obstructive jaundice. We all know that the excretion of bile salts through the urine is irritating to the renal parenchyma and indeed may be associated with a lowering of renal function as shown by reduction in urea clearance. The serum protein was reduced to the so-called critical level at which edema may occur from hypoproteinemia. A reduction of serum protein is

often seen in chronic liver disease, such as cirrhosis or Pick's disease. The calcium is reduced proportionally with the serum protein. The bleeding and clotting studies mean nothing. The stool examination gave a positive guaiac test; with obstructive jaundice, guaiac tests on the stools are often positive in the absence of any neoplasm or large ulceration in the gastrointestinal tract.

The two outstanding findings in this case are obstructive jaundice of high degree and ascites—two rather uncommonly associated findings in cases seen on the surgical service and treated for obstructive jaundice. Furthermore, the picture is colored by the facts that the patient was fifty-eight years old and that her general health was apparently good until five weeks before she came into the hospital. There was a palpable liver, and what was said to be a palpable gall bladder. A significant fact, also, is the absence of chills and fever. Could this patient's disease be primary intrahepatic disease, such as acute hepatitis, frequently called catarrhal jaundice, or perhaps a cirrhosis of the liver with secondary development of hepatoma with compression of the major bile ducts? Both these diagnoses seem to me rather unlikely. The patient's general health had been satisfactory until five weeks before admission. There is no evidence of chronic disturbance in her health prior to that time. The fact that her liver was enlarged, although the upper margin of dullness to percussion is not recorded, would certainly be against the diagnosis of cirrhosis of a type that would produce ascites.

I think we come to a more likely diagnosis when we consider the possibility of common duct stone or carcinoma of the head of the pancreas. In a series of 112 cases of obstructive jaundice of a high degree operated on in this hospital, 34 per cent were due to stone in the common bile duct and 36 per cent to carcinoma of the head of the pancreas or ampulla of Vater. Both these diagnoses have to be thought of here. There is a good deal against the diagnosis of common duct stone. In the first place there is an absence of any history of chronic cholecystitis, an absence of infection and cholangitis, and a complete absence of pain. Stone in the common duct can produce obstructive jaundice without a significant degree of pain, but this is quite unusual. Furthermore, the obstruction in this case is nonfluctuating and complete. These points, it seems to me, taken together make common duct stone an unlikely diagnosis, although possibly no one of these considerations would exclude the diagnosis.

There is a good deal to favor the diagnosis of carcinoma of the head of the pancreas; the age of the patient, the mild symptoms of indigestion, the nausea, the anorexia and the fatigue before the jaundice appeared are consistent. The progressive complete nature of the obstruction, the fact that the gall bladder was palpable, and the presence of enlargement of the liver, all fit this diagnosis, as does the absence of signs of infection. The ascites has to be considered in relation to the jaundice. It would be quite difficult to [illegible] on the basis of common duct stone. On the other hand with obstruction of the bile ducts by carcinoma the tumor might well produce obstruction of the portal vein by involvement of the lymph nodes in the portal fissure, or by direct invasion of the portal vein. So, without spending too much time over it, it seems to me that the most likely diagnosis is carcinoma involving the extrahepatic bile ducts. The question then comes to mind, Was the carcinoma primary in the biliary duct or head of the pancreas, or was it metastatic from a focus elsewhere [illegible]? I do not see that we have any evidence on which to settle this point. The obstruction may be due to carcinoma arising from the ovary or some other intra-abdominal viscus, with secondary involvement of the common duct and portal vein.

I cannot relate the saphenous thrombosis discovered on physical examination to the main disease in this case, although I suppose it is possible that there may have been carcinomatosis of the peritoneum with compression of the iliac veins in the pelvis. I shall have to leave the diagnosis as carcinoma of the extrahepatic biliary duct, probably primary but possibly secondary to a lesion elsewhere in the abdomen. Such a picture as this is occasionally seen in cases with abdominal lymphoblastoma, but we have no evidence on which to make such a diagnosis.

Clinical Diagnosis

Carcinoma of gall bladder with obstruction of common duct.

Dr. Stewart's Diagnosis

Carcinoma of extrahepatic bile ducts, probably primary.

Anatomical Diagnoses

Carcinoma involving common hepatic duct, gall bladder and jejunum, probably primary in hepatic duct, with peritoneal, pulmonary and hepatic metastases.

Icterus.

Arteriosclerosis.

Leiomyomata uteri.

Pathological Discussion

Dr. Tracy B. Mallory: This patient was explored by Dr. Sweet who found a widespread carcinoma throughout the abdomen and noted numerous minute metastases which had practically replaced the liver. The gall bladder was replaced by a solid hard mass of tumor, and at the end of the exploration he thought he was probably dealing with a primary carcinoma of the gall bladder. The patient slowly failed and died in the course of the next couple of weeks.

The autopsy left us somewhat puzzled. We found three masses of carcinomatous tissue, any one of which on gross appearance might have been the primary lesion. The gall bladder was completely filled by tumor masses, the common hepatic duct was completely plugged by tumor, and there was a tumor in the jejunum about 50 cm. below the ligament of Treitz. As I said, any one of the three tumors from the gross point of view could have been interpreted as the primary lesion. The microscopic examination does not carry us much farther. There is cancer in all these areas, but I am inclined to think that neither the gall bladder nor the small intestine focus represents the primary lesion and that the probable primary focus was the common hepatic duct. I admit that there is a good deal of guesswork involved. In ordinary circumstances one would expect that, with a carcinoma of the hepatic ducts above the junction of the cystic ducts, the gall bladder would not have been palpable, but in this case the gall bladder was filled with tumor and therefore was readily felt.

Dr. Stewart: Was there definite involvement of the portal vein?

Dr. Mallory: It was not noted, and it certainly was not occluded. One of its branches may possibly have been invaded enough so that the metastases to the liver had occurred through it. I think it is quite reasonable to assume that there were enough metastases in the liver to have accounted for a severe grade of liver insufficiency. If the portal vein had been obstructed, one would expect the spleen to have been enlarged; it was, however, within normal limits, weighing 230 gm.

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A REPORT ON THE SPECIAL SESSION

The "President's Address" to the New England Surgical Society, which appears in this issue of the *Journal*, is essentially the same as the report given by the author, as senior delegate from Massachusetts to the House of Delegates of the American Medical Association, at the October meeting of the Council of the Massachusetts Medical Society. The address chiefly concerns the propositions of the governmental Technical Committee on Medical Care and the resolutions adopted by the House of Delegates at its special session that define the attitude of organized medicine in regard to the suggested program. Both of these statements are self-explanatory and further comment is unnecessary other than to state that the resolutions of the House of Delegates stand as a chart of guiding principles that will make clear to the representatives of the Government the co-operative spirit of the Association in arriving at some agreement as to the details of procedure in dealing with the health of the nation. The appointment, by the speaker of the House of Delegates, of the committee of nine to confer and consult with proper federal representatives was a necessary and an extremely important step. The interests of the medical profession are in safe hands.

It must be a great relief to all interested parties that out of the welter of controversy of the past few years relating to sociological and medical problems there now appear to be friendly accord and a determination in the profession to carry forward the movement to apply the principles set forth in the resolutions of the House of Delegates of the American Medical Association.

SYPHILIS IN MASSACHUSETTS

The extent of an infectious disease in a community may be surmised or estimated, but figures are needed for the verification of impressions. Much has been written of the presence of syphilis, but few individuals realize that almost 5000 cases are being reported in Massachusetts every year. The report, published in this issue of the *Journal*, of the cases of syphilis and gonorrhea occurring in this state shows that approximately 40,000 cases of syphilis and 53,000 cases of gonorrhea have been reported in the last eight years. During this period 3028 physicians reported cases, but it is difficult to believe that the other 4000 physicians in Massachusetts did not see any cases of syphilis. It is of interest, however, that 1507 physicians reported cases of one or the other disease in 1937, an increase of 30 per cent over the number reporting in 1930. Moreover, a third of the cases were reported by physicians in 1937, in contrast to a quarter of the cases in 1930.

There are other encouraging features in this report. While the total number of reported cases regularly increased during this period, that of early cases decreased so that in 1937 the late cases comprised 75 per cent of the total, in all probability due to the general adoption of routine blood tests and to improved detection. The congenital cases represented only 5 to 8 per cent of the total cases.

No distinction is made as to the early and late congenital cases, but the former should be practically nil if modern diagnostic and therapeutic methods are employed.

The migration of patients to other cities for treatment is an interesting study and indicates the necessity of the private physician's being properly trained for the adequate treatment of syphilis by modern, accepted technics. It is to be regretted that so many patients discontinued treatment prematurely; further efforts by the clinics and by physicians are necessary in the education of the patient concerning the responsibilities of continued treatment for the required time.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

SPONTANEOUS RUPTURE OF THE UTERUS THROUGH A PREVIOUS CESAREAN SCAR

A thirty-nine-year-old gravida III, when thirty-eight weeks pregnant, was admitted to the hospital as an emergency case at 1:00 a. m., October 13, 1926.

The family history was essentially negative. The patient had never been robust, tiring easily, but had not suffered from any diseases except those of childhood. She had undergone a tonsillectomy and an appendectomy. Catamenia began at fourteen and had been fairly regular at twenty-eight-day intervals, lasting four to five days and accompanied by pain on the first day. Her last period began January 25, making her expected date of confinement November 1. Her first pregnancy had been normal throughout and had terminated in a very difficult labor resulting in a stillborn child. Her second pregnancy was terminated by an elective classical cesarean section, with a normal child. Both convalescences had been normal.

During the present pregnancy her blood pressure had ranged consistently from 115 to 130 systolic, and from 70 to 90 diastolic. One urinalysis had shown the slightest possible trace of albumin, but was otherwise negative. Her hemoglobin was 90 per cent (Tallqvist), and the red count 4,850,000. Her pelvic measurements were within normal limits.

The patient was brought six miles to the hospital by automobile and walked from the car into the hospital, doubled up and crying because of severe pain in the lower abdomen. This pain had started suddenly one hour previously and was constantly becoming more severe. The pain was unremitting, not intermittent like labor pains. Examination showed the pulse rate to be 120, the blood pressure 100 systolic, 70 diastolic, and her color pale. On palpation the entire abdomen was exquisitely tender; the uterus could not be definitely outlined, but the fetal small parts were felt directly beneath the skin. The fetal heart rate was 140 and of good quality. There was no external bleeding. The uterus was the size of a full-term pregnancy, and a diagnosis of ruptured uterus was made.

Shortly after admission, the patient became very pale and nauseated, though her pulse did not rise. She was given a ¼ gr. of morphine and intravenous saline, and was prepared for immediate operation. Under ether anesthesia a laparotomy was performed. The baby was found to be lying free in the abdominal cavity, surrounded by a considerable amount of blood and amniotic fluid. The placenta was caught tightly in the ruptured uterine wall, checking marked hemorrhage, and was still sufficiently adherent to the placental site to keep the baby alive. A hysterectomy was performed as the tear in the uterus appeared difficult to repair. At the conclusion of the operation the patient's condition was fairly good. The pulse rate was 140, and the blood pressure 98 systolic, 70 diastolic. She was given 1000 cc. of 10 per cent glucose in saline intravenously. (Today she would have been given a blood transfusion, but at that time transfusions were only rarely given.)

The baby weighed 8 lb., 4 oz., and cried lustily after resuscitation. The patient rallied quickly from the immediate effects of the operation. Her hemoglobin three days postoperatively was 60 per cent (Tallqvist), and the red count 4,000,000. She was started immediately on iron therapy. On the twenty-first postoperative day both mother and baby were discharged well.

Comment. This is another case, before labor, of spontaneous rupture of the uterus in a previous cesarean scar. It is very interesting that although the baby was lying free in the abdominal cavity, the placenta was still attached and the baby alive. The frequency of rupture in cesarean

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

scars has been brought out in these case histories several times. It emphasizes the possibility, and in consequence one cannot too strongly advise that original cesarean sections should never be done unless the indication is a real one, concurred in by expert consultants. One must always realize that no matter who the operator, no matter how smooth the convalescence, cesarean section, whether it be in the lower or the upper segment, may leave a weak scar which may rupture during pregnancy or during labor.

ADDITIONAL COUNCILORS

The following list contains the names of members of the Middlesex South District Medical Society who were elected Councilors at the last annual meeting, but whose names are not included in the "List of Councilors," as published: William B. Bartlett, Clifford L. Derick, Aubrey O. Hampton, Lewis S. Pilcher, Walter N. Secord.

ALEXANDER A. LEVI, M.D., *Secretary*.

DEATHS

ROBERTSON — EWAN A. ROBERTSON, M.D., of 500 Crafts Street, West Newton, died October 18. He was in his seventy-fourth year.

A native of Prince Edward Island he received his degree from the Chattanooga Medical College in 1905. Dr. Robertson studied in England and Germany after which he practiced in Lowell. In 1927 he accepted the post of superintendent of Woodlawn Sanitarium in West Newton.

Dr. Robertson was a member of the American Medical Association, the New England Society of Psychiatry and the Massachusetts Medical Society.

His three sisters survive him.

STETSON — FRANK E. STETSON, M.D., of South Dartmouth, died October 20. He was in his seventieth year.

A graduate of Harvard University, he received his degree from the Harvard Medical School in 1897. He was associated with the Children's Hospital in Boston for a year and later was a member of the staff of St. Luke's Hospital in New Bedford.

Dr. Stetson was a former member of the Massachusetts Medical Society.

His widow, a brother, two sons and a daughter survive him.

MISCELLANY

MAINE NEWS

JOINT MEETING

On October 2 a joint meeting of the editorial board of the *Maine Medical Journal*, the county secretaries and the president and the president-elect of the Maine Medical Association was held at the Penobscot Country Club in Bangor. Dr. F. H. Jackson, chairman of the editorial board, presided.

This meeting had as its purpose a discussion of the best method of securing postgraduate education for the physicians of Maine, and a policy of stimulating interest through the agency of the county societies was outlined by Dr. Frederick T. Hill, chairman of the Committee on Postgraduate Education.

It was proposed that two methods be adopted. The first would be extramural or out-of-state education, where the physician is given instruction in the larger medical centers through the help of the Commonwealth Fund and the Bingham Associates. The second would be made available to physicians intramurally or within the State by the importation of trained teachers, who visit the county medical societies at regular intervals, thus bringing instruction especially to those who are unable, for various reasons, to go outside the State for postgraduate work.

At this meeting the policy of the *Maine Medical Journal* was also discussed, and arrangements made for four stated meetings of the editorial board each year.

FACULTY CHANGES AT HARVARD MEDICAL SCHOOL

A recent notice to the effect that Dr. John E. Gordon had succeeded Dr. Richard P. Strong as professor of tropical medicine at Harvard Medical School was in error. On Dr. Strong's retirement this fall the Department of Tropical Medicine was merged with the Department of Comparative Pathology, and the combined department is now headed by Dr. Ernest E. Tyzzer, George Fabyan Professor of Comparative Pathology.

Dr. Gordon has been appointed professor of preventive medicine and epidemiology at the medical school and the Harvard School of Public Health. This position, held for so many years by Dr. Milton J. Rosenau, has been recently occupied by Col. Frederick F. Russell, who has now been made professor emeritus.

PORTRAIT OF DR. CHRISTIAN

On October 13 the pupils and friends of Dr. Henry A. Christian presented a portrait of him to the trustees of the Peter Bent Brigham Hospital. The portrait was painted by Marie Danforth Page and shows Dr. Christian in his white coat at the bedside of a patient. The ceremony took place in the amphitheater of the hospital, which was filled to capacity.

The presentation address, made by Dr. Elliott C. Cutler, was as follows:

> This occasion is one of those happy moments that arise when all in a community find a common interest. Today we neglect the battle between the stethoscope and the scalpel, and all the superficial trivialities of differing occupations fall aside under the clear light and general devotion to a common purpose.
>
> We are met today to do honor to a man whom we deeply respect and greatly admire, one whose chief devotion is to this hospital, and whose name will travel down the years as part and parcel of our institutional tradition.
>
> Only a few here can know to how great an extent the characteristics and purposes of this hospital, its ideals, its administrative methods and all that we already call its customs and its traditions issue from a single individual. The high purposes to which he set his ambition have brought great credit and renown to the hospital and have played a major role in the good works of those who have sojourned here and then left its doors.
>
> He put the intellectual and spiritual level above other considerations. Selfishness and individual aggrandizement were left aside in a scheme that hoped only for a product in men and ideals that would bring benefit to his profession.
>
> We who have so greatly profited by his high ideals are now met to do him honor, honor such as only pupils and associates can give. His equals and his peers have long since repeatedly honored him in other ways.

Mr. Amory, this portrait is presented by the friends and pupils of Henry Asbury Christian to the Peter Bent Brigham Hospital as another token of their gratitude and esteem. He surely knows that on this occasion our feelings are deeply tinctured by a sincere affection which his kindly spirit has generously allowed us to nourish and enjoy.

Mr. William Amory, chairman of the Board of Trustees, accepted the portrait with many thanks and added another tribute to the great work Dr. Christian had done for the institution.

NOTES

The following teaching and research appointments were recently announced by Harvard University: A. Thornton Scott, Lexington, as assistant in medicine, M.D. Cornell '34; William F. Orr, Jr., Nashville, Tennessee, as assistant in neurology, M.D. Vanderbilt University '35; Joseph Shack, Mattapan, as assistant in physical chemistry, A.B. Harvard '33; Heron O. Singher, Red Hook, New York, as assistant in physical chemistry, A.B. Johns Hopkins University '36; Thomas W. Botsford, Chillicothe, Missouri, as assistant in surgery, M.D. Harvard '35; Timothy J. Kurotchkin, Peking, China, as research associate in bacteriology and immunology, M.D. Imperial University of Tomsk, Siberia '16; Ernst T. von Brucke, Innsbruck, Germany, as research associate in physiology, studied at University of Leipsig; John H. Venable, Emory University, Georgia, as research fellow in anatomy, M.D. Emory University '33; Jonathan F. Meakins, Montreal, Canada, as research fellow in bacteriology, M.D. McGill University '36; William E. Smith, Baltimore, Maryland, as research fellow in bacteriology, M.D. Johns Hopkins '38; Willy K. Rieben, Berne, Switzerland, as research fellow in biological chemistry, M.D. Berne University '38; John Romano, Milwaukee, Wisconsin, as research fellow in neurology, M.D. Marquette University '34; Haim Haimovici, Rumania, as research fellow in physiology, M.D. University of Marseille '37; Per O. Therman, Helsingfors, Finland, as research fellow in physiology, M.D. University of Helsingfors, Finland, '36; Albert Y. Kevorkian, Chestnut Hill, as research fellow in surgery, M.D. Tufts College Medical School '34; William C. Morro, Jr., Fort Worth, Texas, as assistant in physical chemistry, S.M. State University of Iowa '37; Nathan Rudo, Baltimore, Maryland, as research fellow in pathology, M.D. University of Maryland '34; Richard G. Horswell, Chicago, Illinois, as research fellow in medicine, M.D. Northwestern University '36; Ralph B. Miller, Cambridge, as assistant in school hygiene, M.D. School of Medicine of the University of Pennsylvania '30.

Dr. Karl Lowenthal has recently been appointed professor of pathology at the Middlesex University School of Medicine. He received his medical degree from the University of Freiburg in 1915 and was attached to the Department of War Pathology at Metz. He served as assistant at the Pathological Institute, Moabit City Hospital, Berlin, and at the State Institute for Experimental Therapy, Department of Cancer Research, Frankfurt. In 1921 he was associated with the Pathological Institute, Freiburg University, and the Institute for Forensic Medicine, München University. He was head assistant from 1923 to 1926 of the Pathological Institute, Moabit City Hospital, Berlin, and from 1927 to 1929 served as head of the Department of Pathology and Bacteriology of the Public Assurances Hospital Lankwitz, Berlin. He was head of the Pathological Institute and Department of Bacteriology of the City Hospital Lichtenberg, Berlin, from 1929 to 1933. During the past five years Dr. Lowenthal has been professor of histology and embryology at Istanbul University, where he participated in the instruction of pathologic anatomy.

Another new full-time member of the faculty of Middlesex University School of Medicine is Dr. Jacques W. Goldner, professor of histology. Born in Jassy, Rumania, Dr. Goldner studied at Sorbonne, the universities of Paris, Jassy and Cluj and the medical schools of Padova, Turin, Lyon and Jassy, receiving his medical degree in 1921. For the past seventeen years he has been assistant professor at the Histological Laboratory of the medical school of the University of Jassy.

CORRESPONDENCE

STATE LAWS CONCERNING HEALTH EXAMINATIONS BEFORE MARRIAGE

To the Editor: A number of physicians have requested information from this department as to the examination of persons who live or happen to be in Massachusetts and who wish to marry in states which have laws requiring examination for gonorrhea and syphilis before marriage. The laws vary so markedly in the different states that the question of how to proceed is often most confusing, particularly as it pertains to who may take the blood specimen or smear and by what laboratory it must be examined.

We have attempted to abstract the laws of the nine states which have now passed such laws, particularly as they concern physical examination and examination of blood specimens. We have not attempted to abstract that portion of each law which sets forth the time limits between examination and marriage, which vary markedly from state to state. Abstracts of the laws, at least so far as they pertain to these time limits, can usually be found somewhere on the certificate which the physician must sign.

As it happens, it is possible for a Massachusetts physician to make the examination and collect the blood specimen in only a relatively few cases.

The abstracts of the laws are as follows:

CONNECTICUT

Law applies to syphilis only. Physical examination and blood test required. Blood test must be taken, and certificate of freedom from communicable infection must be signed, by a physician licensed to practice medicine in Connecticut. Specimen must be examined at a laboratory in Connecticut, approved by the Connecticut Department of Health. (Rulings as to licensed physician and approved laboratory by Attorney General of Connecticut.) The State Department of Health Laboratory at Hartford is an approved laboratory. Only persons free from communicable syphilis may marry.

ILLINOIS

Law applies to gonorrhea and syphilis. Physical examination, microscopic examination for gonococci and blood test for syphilis required. Only Wassermann and Kahn tests accepted. Blood specimen may be taken and certificate of freedom from infection may be signed by a physician licensed to practice in any state. Specimen, whether smear or blood, must be examined at a laboratory approved by the Illinois Department of Public Health, and that department will accept reports from any laboratory approved by the Massachusetts Department of Public Health. Since the Massachusetts Department of Public Health has not been given legislative authority to approve laboratories in this state, only

the Wassermann Laboratory and the Bacteriological Laboratory of this department are approved. Only persons who are free from infection with gonorrhea and syphilis and have negative microscopic tests for gonorrhea and negative blood tests for syphilis may marry. (Apparently there has been no ruling by the Attorney General of Illinois as to whether a "licensed" physician is one licensed in Illinois, or an approved laboratory one located in Illinois. The law is not specific in these matters. The present interpretation of the law by the Illinois Department of Public Health may, therefore, not be the final one.)

KENTUCKY (effective January 1, 1940)

Law applies to "any venereal disease." Physical examination and "laboratory tests" required. Tests include a Kahn test for syphilis, a dark-field test where indicated or any other recognized test or tests approved by the State Board of Health of Kentucky, and a microscopic test for gonococci when indicated. Blood specimen must be taken and certificate of freedom from infection must be signed by a physician licensed to practice medicine in Kentucky. Specimen must be examined by an approved Kentucky laboratory and must be submitted for examination in a manner prescribed by the State Board of Health of Kentucky. If another state has a law similar to that of Kentucky, of equal requirements as to laboratory tests and doctors' certificates, the certificate of a physician licensed in such a state and examination of specimens at laboratories approved by such a state would be accepted in Kentucky. (Massachusetts has no equivalent law.) Only persons free from infection with any venereal disease may marry.

MICHIGAN

Law applies to "any venereal disease." Physical examination and laboratory test or tests required. Tests include a Kahn test for syphilis, a dark-field test where indicated, and a microscopic test for gonococci where indicated. Blood specimen may be taken and certificate of freedom from infection may be signed by a physician licensed to practice medicine in any state, but specimen must be collected and submitted in a manner approved by the Michigan Department of Health and examined in the Michigan State Laboratory. (No other Michigan laboratories yet approved.) Only persons free from infection with any venereal disease may marry. (The Kentucky and Michigan laws are almost identical, yet their interpretation as to a "licensed" physician is quite different in the two states.)

NEW HAMPSHIRE

Law applies to syphilis only. Physical examination and blood test required. Blood specimen must be taken and certificate of freedom from communicable syphilis must be signed by a physician licensed by the New Hampshire Board of Registration in Medicine. Specimen must be examined at the New Hampshire State Laboratory of Hygiene at Concord. (Assistant Attorney General of New Hampshire has ruled upon meaning of "licensed" physician and "approved" laboratory.) Persons free from infection with communicable syphilis may marry.

NEW JERSEY

Law applies to syphilis only. Physical examination and blood test required. Blood specimen may be taken and certificate of freedom from communicable syphilis may be signed by a physician licensed to practice medicine in any state. Specimen must be examined at a laboratory approved by the New Jersey Department of Health. No laboratories outside of New Jersey have been approved. The State Laboratory is at Trenton. Persons free from infection with communicable syphilis may marry.

NEW YORK (exclusive of New York City)

Law applies to syphilis only. "Such examination, including a standard serological test, as may be necessary for the discovery of syphilis" is required. Blood test may be taken and certificate signed by physician licensed to practice medicine in any state. Specimen must be examined in a laboratory approved by the New York State Department of Health. No laboratories outside of New York State have been approved. Specimen may be sent by air mail to the New York State Department of Health, Division of Laboratories and Research, Albany. Only persons free from communicable syphilis may marry.

NEW YORK CITY

Same as New York State, except that specimen may be examined in any New York State Department of Health laboratory.

RHODE ISLAND

Law applies to tuberculosis, syphilis and gonorrhea. Physical examination and a standard laboratory test for syphilis, approved by the Department of Public Health, required. Blood specimen must be taken and certificate of freedom from communicable infection must be signed by a physician licensed to practice medicine in Rhode Island. Specimen must be examined in a laboratory inspected and approved by the Rhode Island Department of Public Health. Laboratories outside of Rhode Island may apply for approval. (No Massachusetts laboratory, so far as we know, has applied for approval.) Only persons free from infectious tuberculosis, infectious syphilis and gonorrhea may marry.

WISCONSIN

Law applies to "any venereal disease" in the male, but to syphilis only, in the female. Physical examination and the "application of the recognized clinical and laboratory tests of scientific search, when in the discretion of the examining physician such clinical and laboratory tests are necessary" required in males only. Wassermann test for syphilis required for both males and females, but females need not submit to physical examination. Although a physician licensed to practice medicine in any state may sign a statement as to the physical examination of males, only one licensed to practice medicine in Wisconsin may sign the blood test certificate. Specimen may be examined in an "accredited" (not defined) laboratory, but if a Wisconsin physician does not actually draw the blood, evidence will probably be required that it was collected from the person named. Only the Wassermann test is accepted. Only males free from any venereal disease and with negative blood tests, and females with negative blood tests may marry.

N. A. NELSON, M.D., *Director*,
Division of Genitoinfectious Diseases,
Massachusetts Department of Public Health.
State House,
Boston.

THE OSLER EWELME FUND

To the Editor: On August 5, 1937, you generously opened your columns for an appeal for funds to restore the church at Ewelme near Oxford in the name of Sir William Osler. The committee can now report that over $600 was raised and that the money has been gratefully received by the trustees of the Ewelme Almshouse Charity.

HENRY R. VIETS,
For the committee.

Court Lane,
Dedham, Massachusetts.

LICENSE REVOKED

To the Editor: This is to advise you that on September 29, 1938, the license of Dr. R. Reyes Garcia, 353 Dwight Street, Springfield, Massachusetts, was revoked.

STEPHEN RUSHMORE, M.D., *Secretary,*
Board of Registration in Medicine.

State House,
Boston.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of September 6 the following have been accepted:

Gilliland Laboratories, Inc.
Diphtheria Toxin-Antitoxin Mixture, 0.1 L+ (goat)

Jensen-Salsbery Laboratories, Inc.
Antianthrax Serum

Lederle Laboratories, Inc.
Typhoid Combined Vaccine, 20 cc. vial package

Eli Lilly & Company
Ampules Pentobarbital Sodium-Lilly, 7½ gr. (0.5 gm.)
Protamine, Zinc and Iletin (Insulin, Lilly), 80 units, 10 cc.

The National Drug Company
Staphylococcus Toxoid

Parke, Davis & Company
Antipneumococcic Serum (Felton) Type 2, Refined and Concentrated
Soluble Gelatin Capsules Parke, Davis & Company's Standardized Cod Liver Oil, 2.0 gm.

Sharp & Dohme
Protamine Zinc Insulin-Mulford, 80 units, 10 cc.

E. R. Squibb & Sons
Immune Globulin (human) (Placimmunin)
Protamine Zinc Insulin-Squibb, 80 units, 10 cc.

The Upjohn Company
Capsules Ephedrine Sulfate, ⅛ gr. (0.025 gm.)
Capsules Ephedrine Sulfate, ¼ gr. (0.05 gm.)
Ampules Ephedrine Sulfate, ¼ gr. (0.05 gm.)

John Wyeth & Brother, Inc.
Tablets Nicotinic Acid, 50 mg.
Tablets Nicotinic Acid, 100 mg.

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois.

REPORT OF MEETING

EASTERN HAMPDEN MEDICAL ASSOCIATION

A regular meeting of the Eastern Hampden Medical Association was held on October 6 at the Oaks Hotel, Springfield.

The scientific paper was presented by Dr. Charles H. Shamlian and was entitled "Asphyxia: Its prevention and treatment." Dr. Shamlian outlined fifteen main causes of asphyxia, each with many contributing causes. He stated that there are over 50,000 cases of asphyxial death in this country annually, this number being double that due to automobile accidents. All the death certificates, about 10,000, filed with the Springfield Board of Health during the past five years were analyzed and he found that the average annual number of asphyxial deaths is 64 as compared with 21 due to automobile accidents. He then outlined a plan whereby in addition to the usual rescue squads of the police and fire departments and the inhalator crews of the gas and electric companies, trained pneumatological service would be available at all times for an emergency. He decried the high death-rate due to asphyxia and reaffirmed his efforts to reduce it.

A special committee tendered a resolution on the death of Dr. Edward W. Gorman.

J. JOSEPH KLAR, M.D., *Secretary-Treasurer.*

NOTICES

BOSTON DISPENSARY

25 Bennet Street, Boston
Lecture Hall, Second Floor, 9-10 a. m.

MEDICAL CONFERENCE PROGRAM, NOVEMBER, 1938

Tuesday, November 1 — Diagnosis of Internal Derangement in the Knee Joint. Dr. J. D. Adams.
Wednesday, November 2 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, November 3 — Nephritic Clinic. Dr. R. W. Buck.
Friday, November 4 — Rupture of the Lower Lumbar Disk. Dr. W. J. Mixter.
Saturday, November 5 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, November 8 — Progress in Diagnosis and Treatment of Malignant Diseases. Dr. W. M. Shedden.
Wednesday, November 9 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, November 10 — Clinical Problems in Drug Addiction. Dr. Lowrey Davenport.
Saturday, November 12 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, November 15 — Clinicopathological Conference. Dr. Harold Wood and Dr. F. W. White.
Wednesday, November 16 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, November 17 — Medical-Social Case Presentations. Dr. I. Olef and members of Social Service Department.
Friday, November 18 — Some Points in the Management of Heart Failure. Dr. S. A. Levine.
Saturday, November 19 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, November 22 — X-ray Demonstration. Dr. Alice Ettinger.
Wednesday, November 23 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Friday, November 25 — Current Problems in Diabetes. Dr. E. P. Joslin.

Saturday, November 26 — Hospital Case Presentation. Dr. S. J. Thannhauser.

Tuesday, November 29 — The Value of Urethrography in the Diagnosis and Treatment of Lesions of the Lower Genito-Urinary Tract. Dr. M. L. Brodny.

Wednesday, November 30 — Hospital Case Presentation. Dr. S. J. Thannhauser.

GREATER BOSTON MEDICAL SOCIETY

The next meeting of the Greater Boston Medical Society will be held in the auditorium of the Beth Israel Hospital, on Tuesday, November 1, at 8:15 p. m.

Dr. Walter E. Dandy, of Johns Hopkins University School of Medicine and Johns Hopkins Hospital, will speak on "The Diagnosis and Treatment of Lesions of the Cranial Nerves." Drs. Donald Munro, Harry Solomon and Jason Mixter will be the discussers.

CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS, UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Orthopedic Consultant
Haverhill	November 2	Arthur T. Legg
Lowell	November 4	Albert H. Brewster
Salem	November 7	Harold C. Bean
Gardner	November 8	Mark H. Rogers
Brockton	November 10	George W. Van Gorder
Springfield	November 16	Garry deN. Hough, Jr.
Worcester	November 18	John W. O'Meara
Pittsfield	November 21	Francis A. Slowick
Hyannis	November 22	Paul L. Norton
Fall River	November 28	Eugene A. McCarthy

BOSTON GASTROINTESTINAL SOCIETY

There will be a meeting of the Boston Gastrointestinal Society in the Beth Israel Auditorium on Wednesday evening, November 9, at 8:30.

PROGRAM

Intestinal Distention. Dr. Jacob Fine.
Regional Colitis. Dr. Joseph Dubins.
Present Status of Chronic Gastritis. Dr. I. R. Jankelson.
Glucose Metabolism. Dr. B. M. Banks.

Physicians, students and nurses are cordially invited to attend.

FAULKNER HOSPITAL CLINICO-PATHOLOGICAL CONFERENCE

The regular monthly clinico-pathological conference of the Faulkner Hospital will be held on Thursday, November 3, at 5:00 p. m.

Cases will be discussed by Dr. Edward L. Young, Jr., and Dr. William C. Moloney.

DEDICATION OF THE CHEEVER AMPHITHEATER

The new amphitheater in the Dowling Building at the Boston City Hospital will be dedicated to Dr. David Williams Cheever on Monday, November 7, at noon. Mr. Carl Dreyfus, Dr. Fred B. Lund and Dr. David Cheever will speak. Members of the profession are cordially invited to attend the exercises.

JOINT MEETING OF THE BOSTON HEALTH LEAGUE AND THE BOSTON COUNCIL OF SOCIAL AGENCIES

There will be a joint meeting of the Boston Health League and the Boston Council of Social Agencies on Friday, November 4, at 12:15 at the Hotel Sheraton, Bay State Road, Boston. Luncheon will be $1.00 per plate.

Dr. Allan M. Butler will summarize the National Health Conference in Washington last August. Dr. Walter G. Phippen will report on the two recent meetings of the House of Delegates of the American Medical Association. Dr. Channing Frothingham will discuss the recent action of the Council of the Massachusetts Medical Society. Dr. Richard M. Smith, chairman of the Committee for the Improvement of Medical Care, will preside.

All reservations must be in by Wednesday, November 2.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, October 31

Tuesday, November 1

*9-10 a. m. Boston Dispensary. Diagnosis of Internal Derangement in the Knee Joint. Dr. J. D. Adams.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

8:15 p. m. Greater Boston Medical Society. Beth Israel Hospital auditorium.

Wednesday, November 2

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*11:30 a. m. - 12:30 p. m. Psychiatric Conference. Boston City Hospital. Nerve Conference Room, 6th floor, Out-Patient Department.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

Thursday, November 3

*9-10 a. m. Boston Dispensary. Nephritic clinic. Dr. R. W. Buck.

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

5 p. m. Faulkner Hospital, clinicopathological conference.

Friday, November 4

*9-10 a. m. Boston Dispensary. Rupture of the Lower Lumbar Disk. Dr. W. J. Mixter.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Clinical meeting of the Children's Medical Service, Massachusetts General Hospital, Ether Dome.

12:15 p. m Joint meeting of the Boston Health League and the Boston Council of Social Agencies, Hotel Sheraton, Bay State Road, Boston.

Saturday, November 5

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

October 31, November 1, 2, 3 and 4 — Inter-State Postgraduate Medical Association. Page 451, issue of September 22, and page 632, issue of October 20.

November 1 — Greater Boston Medical Society. Notice above.

November 1-30 — Boston Dispensary Medical Conference Program. Page 681.

November 2-28 — Clinics for Crippled Children in Massachusetts, under the Provisions of the Social Security Act. Notice above.

November 3 — Faulkner Hospital. Clinicopathological conference. Notice above.

November 4 — Joint meeting of the Boston Health League and the Boston Council of Social Agencies. Notice above.

November 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

November 5, 1938; February 4, May 15 and 16, 1939 — American Board of Obstetrics and Gynecology. Page 451, issue of September 22.

November 7 — Dedication of the Cheever Amphitheater. Notice above.

November 9 — Boston Gastrointestinal Society. Notice above.

November 10 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

November 15-16 — New England Postgraduate Assembly. Page vii, Advertising Section, issue of September 29.

January 12, 1939 — Peter Bent Brigham Hospital. Clinic conducted by Dr. Christian. Page 633, issue of October 20.

March 27-31 — American College of Physicians. Page 36, issue of July 7.

May 7-15 — International Congress of Military Medicine and Pharmacy. Page 501, issue of September 29.

May 15-19 — American Medical Association, St. Louis, Missouri.

June 6, 7, 8 — Massachusetts Medical Society. Worcester.

September — Boston Psychoanalytic Institute. Page 450, issue of September 22.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH

NOVEMBER 2 — Beverly Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John Homans. Subject: Clinical Forms of Thrombophlebitis with Especial Reference to the Prevention of Pulmonary Embolism.

DECEMBER 7 — Salem Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speakers: Dr. Walter G. Phippen and Dr. Channing Frothingham. Subject: Economic Problems.

JANUARY 4 — Danvers State Hospital. Clinic at 5 p. m. Dinner at 7 p. m.

FEBRUARY 8 — Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Edward Churchill.

MARCH 1 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John Rock. Subject: Endocrinology.

APRIL 5 — Addison Gilbert Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Ethan Allan Brown. Subject: Allergy.

MAY 10 — Annual meeting. Salem Country Club, Peabody.

HAMPDEN

NOVEMBER 3 — Censors' meeting. Rooms of the Academy, Springfield.

SUFFOLK

NOVEMBER 3 — Censors' meeting. 4 p. m., Boston Medical Library.

NOVEMBER 30 — Program of clinical and investigative activities. Staff of the Massachusetts General Hospital, Boston Medical Library, 8:15 p. m.

JANUARY 25, 1939 — Symposium on Diabetes. Dr. Elliott P. Joslin and associates, Boston Medical Library, 8:15 p. m.

MARCH 29 — Joint meeting with New England Pediatric Society, Boston Medical Library, 8:15 p. m. Program and speakers to be announced.

APRIL 26 — Annual meeting in conjunction with Boston Medical Library, at 8:15 p. m. Election of officers. Program and speakers to be announced.

BOOKS RECEIVED FOR REVIEW

Handbook of Physiology and Biochemistry. W. D. Halliburton and R. J. S. McDowall. Thirty-fifth edition. 973 pp. Philadelphia: P. Blakiston's Sons & Co., Inc., 1937. $5.50.

Dr. Colwell's Daily Log for Physicians: A brief, simple, accurate financial record for the physician's desk. Champaign: Colwell Publishing Co., 1938. $6.00.

Studies from the Rockefeller Institute for Medical Research. Volume 108. 595 pp. New York: The Rockefeller Institute for Medical Research, 1938. $2.00.

Human Pathology: A textbook. Howard T. Karsner. Fifth edition. 1013 pp. Philadelphia and London: J. B. Lippincott Co., 1938. $10.00.

One Hundred Years of Publishing, 1837-1937. 83 pp. Boston: Little, Brown & Co., 1937.

The Medical Applications of the Short Wave Current. William Bierman. 379 pp. Baltimore: William Wood & Co., 1938. $5.00.

A Synopsis of Physiology. A. Rendle Short and C. L. G. Pratt. Third edition. 325 pp. Baltimore: William Wood & Co., 1938. $3.50.

The Rockefeller Foundation. International Health Division. Annual Report, 1937. 243 pp. New York: The Rockefeller Foundation, 1938.

The Practice of Medicine. Jonathan C. Meakins. Second edition. 1413 pp. St. Louis: The C. V. Mosby Co., 1938. $12.50.

Biology and Pathology of the Tooth and Its Supporting Mechanism. Bernhard Gottlieb and Balint Orban. 195 pp. New York: The Macmillan Co., 1938. $5.00.

Mental Conflicts and Personality. Mandel Sherman. 319 pp. New York, London and Toronto: Longmans, Green & Co., 1938. $2.25.

BOOK REVIEWS

Practical Otology. Morris Levine. Second edition, thoroughly revised. 416 pp. Philadelphia: Lea & Febiger, 1938. $5.50.

This book is a second edition of a compilation of lectures to students of otology. It is based essentially on the personal experience of the author and hence omits controversial subjects and operative procedures not subscribed to by him. It is a book more valuable to the practicing otologist and postgraduate student in otology than to undergraduates or the general practitioner, for whom the technicalities of operative procedures can have little applicability.

The newer subjects of petrositis and allergy in aural disease have been brought up to date, and the chapter on otitic meningitis has been rewritten.

The arrangement of the book is a practical one for the physician with a good working knowledge of otology. The illustrations are in general helpful and up to date, though the temptation to reproduce cuts from outmoded textbooks has not always been resisted.

The practicing otologist will find in this volume a concise description of such operative procedures as have proved satisfactory in the author's experience and a helpful guide in the treatment of all the otologic complaints with which he is likely to come in contact. Theory and records of experimental work of interest to the research man in the laboratory have wisely been omitted.

Sulfanilamide Therapy of Bacterial Infections: With special reference to diseases caused by hemolytic streptococci, pneumococci, meningococci and gonococci. Ralph R. Mellon; Paul Gross and Frank B. Cooper. 398 pp. Springfield, Illinois, and Baltimore: Charles C Thomas, 1938. $4.00.

This text is of interest because of the increasing attention which is being paid to the sulfanilamide compounds in the treatment of infections. At first sight the book appears to be a comprehensive review of most of the literature, but it lacks systematic treatment of the special fields in which sulfanilamide is employed and appears to be a grouping of abstracts from the literature rather than an organized tableau of the subject.

The book is to be recommended primarily to pharmacologists and pathologists who are interested in the field of chemotherapeutic agents.

The statements referring to priority in the use of sulfanilamide in this country by the authors, in contradistinction to the investigations of Long and Bliss, seem needless to the reviewer.

A Textbook of Histology. E. V. Cowdry. Second edition. 600 pp. Philadelphia: Lea & Febiger, 1938. $7.00.

Histology today is a vigorous, constantly growing member of the medical sciences. Contributions from the fields of biochemistry, physiology, physics, pathology and clinical medicine enrich the interpretation of its factual content. Professor Cowdry has admirably succeeded in correlating this material in the thoroughly revised second edition of his textbook. It will remain a pacemaker in the field of histology. The aim of the author is high, and the subject matter has been deliberately planned for the best minds. Such an approach presupposes adequate preliminary training, a demand which should be made by the altogether too complacent histologists. A strong case could be made out for the wide use of the book as a text if this inequality in preliminary training were satisfactorily solved.

The book contains thirty-three chapters arranged under the functional headings: "Water: The essential vital me-

dium"; "Blood: The principal integrator"; "Absorptive Drainage into Blood"; "Chemical Integration by Endocrine Products in Blood Stream"; "Intake of Water"; "Nutriment, Accessory Food Factors and Removal of Waste"; "Oxygen Consumption and Carbon Dioxide Elimination"; "Regulation of Constitution of Blood and Removal of Waste"; "Rapid Neural Integration in Response to Internal and External Stimuli"; "Architectural Support"; "Movement"; "Perpetuation of the Race"; and "Unification, Protection and Adjustment." It is not to be supposed that morphological considerations are subordinated to the above physiological arrangement of topics.

An unconventional but most useful introduction has been added which deals with certain highlights in the history of histology, but one misses the names of Karl Ernst von Baer and Marie-Francois-Xavier Bichat. Attention should be drawn to the excellent chapters devoted to introducing the endocrines and the digestive system. Many new illustrations bring out the normal variations in structure upon which depend degrees of physiological activity, including several showing the histologic changes in the process of aging.

Students and teachers will find their concept of histology sharpened; they will find themselves facing the subject "in the growing zone," as Professor Sabin so aptly stated, once this stimulating and provocative book is seriously taken in hand. In the last analysis this new venture will be judged in accordance with the dictate of Roger Bacon: *"Sine experimentia nihil sufficienter sciri potest."*

The Form and Functions of the Central Nervous System. Frederick Tilney and Henry A. Riley. Third edition. 851 pp. New York: Paul B. Hoeber, Inc., 1938.

Five years have passed since the second edition of this book, during which time both the senior author and the publisher have died. The junior author and the publisher's son have now presented a third edition, much of the work having been done, however, before the death of either Dr. Tilney or Mr. Hoeber. This book has found a welcome among neurologists for it is well recognized that Dr. Tilney was an accomplished neuroanatomist and Dr. Riley a competent neurologist. The weakness of the book, a defect that still persists, lies in the fact that neither was a neurophysiologist. The anatomical part of the book offers no criticism, but the physiology is far out of date, and a book has recently appeared which contains much material of importance not found in this volume. With this fact in mind, the book is considered as a sound text — perhaps as useful as any book in the field — for quick reference to anatomical details. The physiology — sound so far as it goes — cannot be relied on, for the authors have failed to keep up with the rapidly accumulating, newer knowledge of the functions of the nervous system.

The History of Bacteriology. William Bulloch. 422 pp. London, New York and Toronto: Oxford University Press, 1938. $3.75.

This book is based on a series of lectures given at the University of London in 1936. The author, a well-known bacteriologist, has long been interested in the subject. He wrote the history of bacteriology for a system of bacteriology, published in 1930. Dr. Bulloch now presents for the first time a complete account of a relatively new branch of medicine. The development is taken up in chronological order, fully illustrated and annotated by photographs of individuals, reproductions of laboratory apparatus and an extensive bibliography. In addition, brief biographical notices of some of the early workers in bacteriology record data not found in any other publication. The history, beautifully written, at once becomes a standard text on the subject.

You Can Sleep Well: The A B C's of restful sleep for the average person. Edmund Jacobson. 269 pp. New York and London: Whittlesey House, McGraw-Hill Book Co., Inc., 1938. $2.00.

This is a book for laymen by an enthusiast on the subject of "relaxation." Backed up by a certain amount of physiological evidence, the author has expanded his theme in a way that cannot help but be distasteful to the thoughtful physician. The theories that muscular tension is connected with sleep and that "relaxation," or loss of not only voluntary movement but "residual tension," as the author puts it, is necessary before sleep can be attained are not widely accepted by either physiologists or psychologists. On a weak base, Jacobson tries to build a heavy structure.

The Troubled Mind: A study of nervous and mental illnesses. C. S. Bluemel. 520 pp. Baltimore: The Williams & Wilkins Co., 1938. $3.50.

This is a sound, personal book on nervous and mental diseases by a physician in a private sanatorium in Denver, Colorado. Written in an easy style, adapted to the nonmedical reader, as well as students, the whole field of the psychoneuroses and psychoses is covered. There are many practical points brought out by the author, whose long experience in handling mental and nervous patients gives him an exceptional background for his exposition of the subject. Particular attention is paid to the relation of mental disease to crime, politics, religion and war. The book is an excellent study by a scholarly author.

Aids to Histology. Alexander Goodall. Fourth edition. 151 pp. Baltimore: William Wood & Co., 1938. $1.25.

Students of biology as well as medical students will welcome this useful pocket type of book as a quick review in preparation for examinations in this subject. The text is easily readable and will undoubtedly stimulate students to dig deeper into textbooks which give more detailed discussions of structures considered. There is a valuable chapter on histological methods which all students should read carefully.

A College Textbook of Pharmaceutical Botany. Heber W. Youngken. Sixth edition. 793 pp. Philadelphia: P. Blakiston's Son & Co., Inc., 1938. $4.75.

Here is a book which is well calculated to be widely used either as a text or as a book for collateral reading by both teachers and students in universities or professional schools giving courses in pharmaceutical botany. This new edition presents a broad general treatment of botany, with particular stress on histology, cell contents, taxonomy and medicinal plants. In order to bring the subject matter up to date, new chapters have been added on "The Living Cell," "The Normal Protoplasmic Cell Contents" and "Genetics and Evolution." The author gives a good account of the microscope and of certain phases of histological technic. The type is large; the illustrations and photographs are clearly delineated, with 121 new figures added since the last edition.